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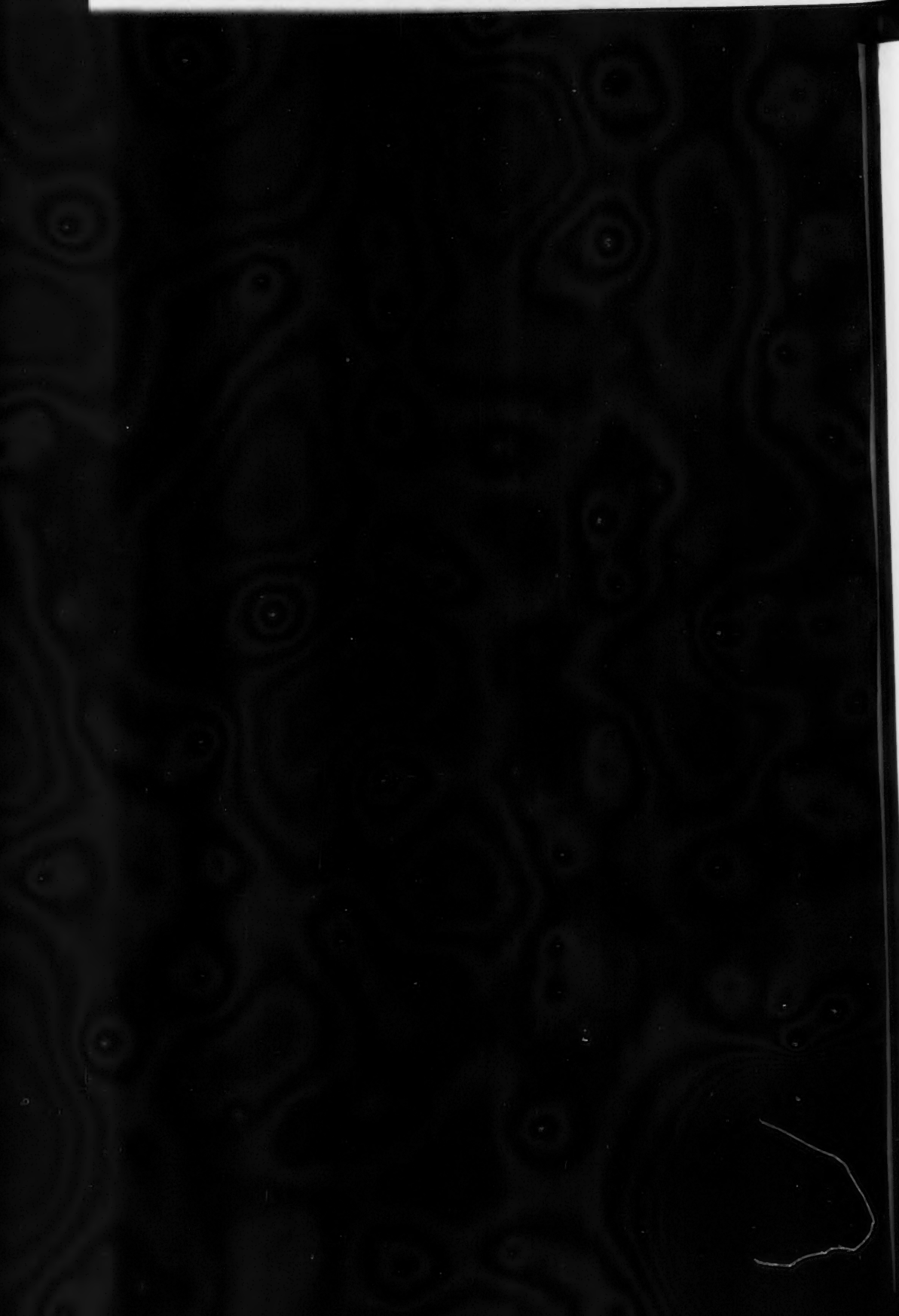

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CURRENT STATUS OF THE TREATMENT OF PULMONARY TUBERCULOSIS

D. B. RADNER, M.D.*

As the result of over emphasis of the declining death rate in tuberculosis, prevalent in the lay and professional press, a form of complacency regarding this particular communicable disease has developed throughout this country. This is not meant to imply that the rapidly declining death rate is to be viewed with alarm or to be decried. In reality, the death rate from tuberculosis has been steadily declining since the turn of the century, with a more precipitous fall during the past decade. Deaths from tuberculosis have declined 57 per cent during the past five years alone. The provisional estimate of mortality from tuberculosis during the year 1954 is represented as being between 10 and 12 per 100,000 population. This fact alone leads some to believe that tuberculosis is a rapidly disappearing disease. On the other hand, national statistics show that the newly reported cases of tuberculosis have declined only slightly, estimated to be only about 3 per cent per year. In 1954, there were 86,000 new cases of tuberculosis reported to the health authorities in this country. The United States Public Health Service states that there are 1,200,000 people with, active or inactive, tuberculosis in the United States today. Of this group, 250,000 are active cases under some form of treatment, either in hospitals or at home. In addition, there are estimated to be at least 150,000 active cases, as yet, unknown to the Health Departments. Of the known 250,000 active cases, less than one-half are receiving some form of institutional care and less than 40 per cent are receiving any drug therapy. Approximately 18 per cent of these people are receiving no medical care of any form. One need not employ complicated mathematical formulae to recognize that if the newly reported cases number more than four times the number of deaths per year, that a tremendous reservoir of cases of tuberculosis is developing in this country year by year. Our health authorities estimate that about 50,000,000 people in this country have a positive tuberculin reaction indicating the presence of at least a primary infection. On the basis of current statistics, it should be appreciated that the greatest decline in mortality has occurred in the young adult age groups even though the level of newly reported cases remains fairly high for this group. The newly reported cases and mortality remain high in the older age groups. The non-white races have a mortality almost three times as high as that of the white race. Male rates are approximately twice those of the female rate, with the difference particularly marked among the white groups. The white males have a mortality rate almost three times that of the white female

*Associate Clinical Professor of Medicine, The Chicago Medical School; Director, Chest Department, Michael Reese Hospital, Chicago, Illinois; Chairman, Chest Department, Mt. Sinai Hospital, Chicago, Illinois; Medical Director, Winfield Hospital, Winfield, Illinois.

race. Lest one be deluded into thinking that most of these newly reported cases are of no clinical significance, it should be pointed out that more than one-third of these cases have positive sputa and 78 per cent of the newly reported cases are in the moderate or far-advanced stage of the disease.

The statement is frequently made that tuberculosis is a different disease from what it was in the past. Speculation, as to whether or not the tubercle bacillus has lost some of its virulence is heard in many quarters. Clinicians have pointed out that the disease appears to be milder clinically with fewer instances of intestinal and laryngeal involvement than seen in the past. It is not difficult to see that such impressions coupled with a sharply declining death rate would lead to such speculation. If these statements are true, then there are three possible explanations: (1.) the tubercle bacillus may have lost some of its virulence; (2.) the host may have increased resistance; (3.) there may be some change in the conditions under which the bacilli reach the host. From the evidence to date, there is no substantiation of the first thesis. Bacilli obtained from fresh untreated cases of pulmonary tuberculosis injected into the guinea pig produce the same disease as of old. Many of the organisms considered saprophytes in the past are now recognized as pathogens. In all likelihood, the changed picture in tuberculosis epidemiology is due to increased host resistance and a change in the circumstances under which the infection develops. The increased resistance in man is not considered to be the result of genetic change, but rather to the marked improvement in nutrition during the past twenty-five years, in this country. Furthermore, because of attempts to isolate, segregate, and treat open cases of tuberculosis, a smaller number of organisms are to be found in any community in a position to invade the uninfected. The partial isolation and diminished number of tubercle bacilli in a population, which is now enjoying an improved nutritional state in a society of high living standards is more likely the explanation of the clinician's observation.

From the above, it becomes apparent that tuberculosis is still the greatest killer among the communicable diseases. It is still a threat to life to the age groups from birth to three years, in adolescence especially in the female, and in the elderly male. The number of infected individuals will continue to rise in the population for some years to come. These individuals must be treated when in the active state, and the survivors will require continued uninterrupted medical supervision in order to prevent and to treat a relapse of the infection. Even under the most ideal circumstances, treated cases of tuberculosis may break down and become active again. With the increased span of life, which the American public now enjoys, there will be ample opportunity for the quiescent and arrested case of tuberculosis to relapse under the stresses and strains of this prolonged life. The individual who has been infected with tuberculosis must be protected, with adequate therapy, against such future hazards of breakdown. The community must also be protected against the breakdown of the individual's disease, lest the population be infected at a persistently high rate. If we are to reduce the newly reported cases of tuberculosis from its present high rate, both the medical profession and the public at large must be educated to the necessity for therapy and isolation of the open active cases of tuberculosis. Despite almost fifty years of intensive educational effort to get the public to accept treatment for *open* tuberculosis, our figures show that about 40 per cent of patients admitted to tuberculosis hospitals still leave, against medical advice, before therapy is concluded. These partially treated cases act as a reservoir of infection in the community and because of the declining death rate will continue to do so for many years to come. Many of these people appear clinically well, even though they discharge tubercle bacilli in the sputum daily. In addition, interruption of treatment has resulted in inadequately healed pulmonary lesions, which present greater problems in treatment often requiring hazardous radical therapy later in life. It is most desirable

that the physician, who is first to discover tuberculosis in the patient, should be realistically oriented in the significance of the infection and in the proper management of this process.

CRITERIA OF ACTIVITY

The absence of symptoms does not rule out an active pulmonary process. The majority of early active cases of pulmonary tuberculosis do not manifest clinical symptoms. The criteria for active disease include in addition to symptoms, both radiographic and bacteriologic evidence. In addition to the history and physical examination, the chest x-ray and bacteriologic studies of sputum offer the physician the greatest help in determining when activity of the pulmonary disease is present. In general, radiologic evidence of a pneumonic or cavitary lesion or a changing appearance in the lesion over serial studies indicates radiologic activity. This is true, whether the disease is regressive or progressive. However, radiologic stability is no assurance of inactivity. Many patients will demonstrate radiologically stable disease over years of time, while they are bacteriologically positive throughout. Bacteriologically, the presence of acid-fast bacilli in the sputum on direct smear or concentrates, confirmed by culture or animal inoculation, is absolute evidence of an *active* process. Unfortunately, many patients do not discharge bacilli in large enough numbers in order to make this finding easily obtainable before extensive and advanced destructive disease is present. Minimum bacteriologic studies should include five concentrate examinations, of which at least three should be cultured or inoculated into animals. In the presence of symptoms or radiologic instability, treatment may be begun before culture or animal inoculation reports have returned. In the absence of radiologic evidence of acute disease or destructive lesions in a patient without clinical symptoms, negative bacteriologic reports do not permit the clinician to ignore the pathologic process. Radiographic and bacteriologic studies should be repeated at frequent intervals over a period of at least three months before the disease may be classified as *arrested*. The presence of acid-fast bacilli in the sputum should prompt the clinician to isolate and begin treatment of the patient immediately.

TREATMENT

For many decades prior to 1944, the successful treatment of active pulmonary tuberculosis consisted of an extended period of bedrest. In more recent times, more specific attempts at treatment included reversible collapse procedures, followed more recently, by surgical collapse methods. With the rapid strides made in surgery most recently, and as the result of the control of infection, hemorrhage, and shock, together with improved methods of anesthesia, resectional surgery came into prominence. All of these methods, when carried out meticulously by the experienced physician, resulted in a high degree of success in controlling the active disease process. However, these methods of therapy were less successful in preventing subsequent relapse. Prior to the use of the present antimicrobial agents the relapse rate of treated cases ranged between 28 and 30 per cent. Twenty-five years of experience in the treatment of active pulmonary tuberculosis using rest, collapse and surgical procedures, led to a better understanding of the use of each of these procedures, as well as their limitations. Since 1944, the development of more specific antimicrobial agents has led some physicians to discard the older methods of therapy. Most authorities[1,2] today are of the opinion that the experience, to date, with the new antimicrobial agents does not, as yet, permit us to discard the previous successful methods of treatment. Rather, it is the attempt of most physicians experienced in the treatment of tuberculosis to seek the proper combination of new and old methods most suitable to control the infection in the shortest possible time with a minimum loss of functioning tissue. In addition to the above, the therapeutic aim should include the minimizing of all future relapse possibilities.

Bedrest

Bedrest is still indicated in the treatment of the active pulmonary tuberculous lesion together with the aid of the

antimicrobial agents, which exert powerful bacteriostatic effects on the tubercle bacillus. The extreme forms of bedrest have been modified. Furthermore, with the more rapid defervescence of fever, the earlier reduction in cough and expectoration, and the more rapid conversion of sputum from positive to negative, the duration of bedrest has been shortened. It is the opinion of the majority of phthisiologists, that the treatment of active pulmonary tuberculosis should include bedrest for varying periods of time, depending upon the extent and the nature of the disease. It is still considered desirable, that most cases of active pulmonary tuberculosis have therapy instituted in a tuberculosis hospital. This is especially true of the case with both cavitation and positive sputum. The average length of hospitalization has been materially shortened since the introduction of the newer antimicrobial agents. The most recent figures indicate that the average period of hospitalization in private sanatoria is six months; that in the public tuberculosis hospital, twelve months; while the average stay in the Veterans Administration Facility is eighteen months. The hospital environment has the advantage of isolation of the open active case, the education of the patient and affords an excellent opportunity for the rehabilitation of the patient. With the exception of highly selected private patient groups, ambulant drug therapy of open cases of tuberculosis has not been nearly as successful either in the control of active disease or the prevention of relapse, as the hospital treated groups.

Antimicrobial Agents

The most widely used antimicrobial agents in the treatment of tuberculosis today are streptomycin (SM), dihydrostreptomycin (DHSM), para-amino-salicylic acid (PAS), and isoniazid (INH). Streptomycin was discovered in 1944 by Dr. Selman Waxman and to this date is still considered to be the most potent of the antibiotic agents available. During the first years of trial, it became apparent that streptomycin was most dramatically effective in miliary tuberculosis without meningitis, tuberculous pneumonia, tuberculous laryngitis, enteritis, and mucous membrane lesions. Since these forms of tuberculosis were responsible for a high mortality and morbidity rate, streptomycin was received with wide acclaim and dramatic excitement. With experience in the administraiton and effects of streptomycin, both the dosage and duration of treatment underwent considerable change. The limitations of streptomycin therapy were soon apparent following the development of neurotoxicity, chiefly that of damage to the vestibular branch of the eighth nerve. Dihydrostreptomycin was developed in an attempt to obviate this neurotoxicity but experience taught that this antibiotic also led to damage—to the auditory branch of the eighth nerve. By diminution in dose, neurotoxicity has been considerably reduced. The present dosage most widely employed is 1.0 Gm. intramuscularly twice weekly. This dose has been found to be of adequate therapeutic value in all cases of tuberculosis except for the miliary and meningeal forms where the same dose is employed daily. In meningeal tuberculosis, doses as high as 2 Gm. daily are employed by some. Streptomycin or dihydrostreptomycin are practically never employed alone but are generally combined with another of the antimicrobial agents. Recently Hinshaw and his co-workers[3] reported on the use of a mixture of equal parts of streptomycin and dihydrostreptomycin to reduce eighth nerve toxicity. They noted that the mixture reduced, but did not eliminate eighth nerve toxicity. Since eighth nerve toxicity is encountered with relative infrequency using one gram twice weekly many physicians have not considered it necessary to employ the combination known as *Streptoduocin*. It is imperative, however, that all patients receiving dihydrostreptomycin or streptomycin have audiometric and caloric tests done at three month intervals to detect eighth nerve toxicity as early as possible.

Para-amino-salicylic acid was introduced in 1946 by Lehman. While this compound was found to be distinctly inferior in potency to streptomycin, it has been widely employed since its introduc-

tion. The therapeutic dose requires twelve grams of para-amino-salicylic acid daily. While Lehman recommended a dose of 15 grams per day, and others, in England and Europe, have found it possible to give 20 to 25 grams per day, most American physicians have found it difficult to administer more than 12 grams per day. In addition to toxic effects, such as drug fever, dermatitis and purpura which occur in a smaller percentage of patients, the daily administration of the therapeutic dose leads to anorexia, flatulence and diarrhea in a larger percentage of patients. The combined use of twelve grams of PAS daily with streptomycin, one gram twice weekly, soon led to the discovery that the PAS was of intrinsic value in extending the period of effective streptomycin therapy, by delaying the emergence of resistance. Thus, combined therapy with streptomycin and PAS, by permitting a longer period of effective treatment, resulted in a higher percentage of sputum conversions. It also resulted in a larger number of patients being adequately prepared for surgical intervention. With the continued employment of a combined streptomycin and PAS regime, it was soon learned that the use of one drug alone, for even short periods of time, encourages the development of bacillary resistance to that drug. Streptomycin resistance was usually found to be permanent. PAS resistance was not considered to be permanent, and after periods of time the organisms, in many cases, again became sensitive to PAS. The development of bacillary resistance was definitely correlated with the persistence of cavities in the lung.

In 1952, isonicotinic acid hydrazide—now known as isoniazid—was introduced as the newest and most potent of the chemotherapeutic agents in the treatment of tuberculosis. Isoniazid has been found to be relatively non-toxic and of higher potency than PAS. It is, approximately, of the same bacteriostatic potency as streptomycin. Unfortunately, when employed alone, resistance to isoniazid develops rapidly, appearing in some instances as early as the fourth or sixth week of treatment. It is employed in doses of 4 to 5 mgm. per kilo of body weight, averaging 150 mgm to 300 mgm daily for an adult. In doses of 300 mgm per day and especially in higher doses, it has been found to produce acute urinary retention, obstipation, mild mental changes characterized as euphoria, loss of memory, nightmares, inability to concentrate, visual and auditory hallucinations and, occasionally, paranoid psychoses[4]. More commonly, and of greater significance, is the development of peripheral neuritis which can be prevented by the administration of pyridoxine. Once peripheral neuritis is present, pyridoxine appears to be of little benefit.

Clinical and laboratory experience with the above three antimicrobial agents has led to a better understanding of their proper use. It is now recognized that with the exception of rare instances, it is best to use a combination of two antimicrobial agents in uninterrupted administration for a minimum of twelve months and from 18 to 36 months in others[5]. In far advanced chronic cases, many investigators are now employing combinations of antimicrobial agents for an indefinite period. Studies conducted by the United States Public Health Service and the Veterans Administration Facilities indicate that combinations of streptomycin and isoniazid, and isoniazid and PAS are of equal value, when measured by their ability to produce radiologic clearing, conversion of sputum, closure of cavity, reduction of symptoms and weight gain. It appears that streptomycin and PAS are slightly inferior as a combination therapy in some of these respects. Some authorities hold the opinion that the simultaneous use of streptomycin and isoniazid is less desirable because of the possibility of resistance to both of these potent agents developing. It is their opinion that either streptomycin or isoniazid should be withheld for future use in case of relapse or as coverage during and following thoracic surgery. In some hospitals, streptomycin and PAS are employed throughout the period of hospitalization and are followed by isoniazid and PAS during the post-hospital continuation of therapy. This avoids the necessity of intramuscular injections after discharge from the hospital.

While current opinion recommends employment of combined antimicrobial therapy in all instances of active pulmonary tuberculosis, the actual duration of therapy recommended is less uniformly agreed upon. The minimum course of uninterrupted therapy is considered by most to be twelve months. Tuberculous pleurisy with effusion, should receive eighteen to twenty-four months of therapy as a minimum. In general, it is the consensus that antimicrobial therapy should be continued for approximately six months after reaching the "target point." The target point is considered to be that point at which cavity closure, maximum radiologic stability, and conversion of sputum from positive to negative are obtained. A recent study evaluating the long term results, after ten months of combined therapy, indicated a relapse rate of 15 per cent after three years. In a more recent study, by the same group, extending the duration of therapy to two and three years, the relapse rate was reduced to less than 2 per cent. The latter group included 18 per cent of patients, who had resectional surgery performed during antimicrobial treatment[6].

Other antimicrobial agents more recently employed and of some value are viomycin, pyrazinamide and most recently cycloserine. Viomycin is much less effective and more toxic than either isoniazid or streptomycin. It is employed in doses of 2.0 Gm. intramuscularly twice weekly. It produces renal irritation and disturbed electrolyte balance with a lowering of the phosphate, potassium and sodium in the serum[3]. It has also been found to be neurotoxic. There are occasional patients who can be tided over critical periods in their disease, or whose organisms are resistant to the other antimicrobial agents, who may benefit from viomycin therapy. Pyrazinamide (PZA), when employed in combination with isoniazid, in experimental tuberculosis in mice, is reported to eradicate tuberculosis infection in animals treated for three months[7]. More recent reports on this combination of drugs, used in human patients, indicate moderately severe liver toxicity[8]. Isoniazid resistance is apparently delayed by the concommitant administration of pyrazinamide. However, because of its toxicity, further studies should be made with this compound before its promiscuous use in humans is recommended. Cycloserine is the most recent of the antibiotic substances used in the treatment of tuberculosis[9]. This compound appears to have an antimicrobial activity in tuberculosis, in man, which could not have been anticipated from the *in-vitro* and *in-vivo* laboratory studies. Preliminary clinical reports using 1.0 Gm. of cycloserine daily (orally), indicate that it has the ability to halt the progress of streptomycin-sensitive as well as streptomycin resistant disease. This antibiotic appears readily in all body fluids and results in a high degree of clinical improvement, less striking x-ray clearance and sputum conversion. However, it is much too early, in the experience with cycloserine, to state what its place in tuberculosis therapy will ultimately be. It is not too soon to state that central nervous system toxicity is quite apparent from the early reports.

SURGICAL THERAPY

Collapse therapy, in the treatment of pulmonary tuberculosis, enjoyed wide popularity in the interval between the first and second World Wars. This was the period of active therapy, prior to the introduction of the antimicrobial agents. The most widely used form of collapse therapy was artificial pneumothorax. During the past twenty years artificial pneumoperitoneum gained increased favor as a form of collapse therapy. Pneumothorax lost favor with many, because of the high incidence of complications that developed, especially tuberculous empyema, atelectasis and a permanently un-expanded lung. Even in the successfully re-expanded cases it was soon learned that severe encroachment on pulmonary function resulted from pneumothorax therapy. While artificial pneumoperitoneum avoided the more serious complications of pneumothorax it was found to be less effective in cavity closure and frequently led to impairment of diaphragmatic function, when continued for a long period of time. Disturbance of

diaphragmatic function was most commonly encountered in cases where pneumoperitoneum was combined with phrenic nerve interruption. While artificial pneumothorax is employed relatively infrequently today, there are some treatment centers in this country that still employ artificial pneumoperitoneum without phrenic nerve interruption, in combination with drug therapy. It is the opinion of those who continue its use, that it is of value in bilateral disease, in stabilizing the diseased areas and preparing the patient for surgical intervention more readily. The use of pneumoperitoneum as a definitive form of therapy has lost considerable favor.

Permanent collapse procedures which began with the classical thoracoplasty more than twenty years ago, has saved many lives in the past and has resulted in an inactive, sputum-negative disease in approximately 80 per cent of the patients so treated. More recently, the classical thoracoplasty has given way to a modified collapse procedure employing paraffin, oil or one of the synthetic plastic materials as a prosthetic agent. This has permitted a more selective collapse with less encroachment on normal lung and a marked reduction in post-operative scoliosis. The modified thoracoplasty with plombage is performed in a single stage, as compared with the two or three stage operation employed in the classical thoracoplasty of the past. The modified thoracoplasty is still employed where resectional surgery is inadvisable, because of the extent of the disease or where resectional surgery is contraindicated for other reasons. The thoracoplasty procedure is ineffective in the presence of cavities more than 4.0 cm in diameter or in the presence of broncho-stenosis.

Resectional surgery in pulmonary tuberculosis[10, 11, 12, 13] came into being with the advent of antibiotics. With the control of active infection and the fibrous walling-off of caseous and cavitary disease, techniques were developed for the resection of destroyed areas of lung, residual cavities and those portions of the lung drained by the destroyed bronchi. Disease confined in one broncho-pulmonary segment can be successfully removed by segmental resection. Lobectomy is employed where the diseased process has crossed the segmental plane and involves all the segments of the lobe. Lobectomy may also be employed where the lobar bronchus is completely destroyed, even though all the segments of the lobe may not be involved. In general, resectional surgery is employed after maximum resolution of the disease is obtained with bedrest and combined uninterrupted antimicrobial therapy. Medlar[14] pointed out that the residual caseous focus, persisting after drug therapy, was the source of future relapse and reactivation. Many of these residual closed necrotic foci which persisted under combined antimicrobial therapy, were resected under protection of continued drug therapy given for extended periods of time post-operatively. It was soon found that the majority of these necrotic foci contained viable tubercle bacilli. The hazard to future breakdoown in residual foci more than 2.0 cm in diameter was apparently correlated with the persistent patency and healing of the broncho-cavitary junction[15, 16]. This permitted expulsion and evacuation of the necrotic contents from the tracheobronchial tree at some future date. When viable bacilli were present in the necrotic material, extension and reactivation of tuberculosis ensued. With widespread use of this resection technique, large scale studies of the resected material were accumulated and it was found that while acid-fast bacilli were present in great numbers in this necrotic material difficulty was encountered in culturing these organisms and in reproducing the disease in experimental animals. More recently, Hobby[17] reported on specialized methods of culturing organisms from these foci, indicating that the organisms were still viable. Further, while the organisms in some instances, especially after isoniazid therapy, lost their ability to produce tuberculosis in guinea pigs, they were found to be pathogenic for certain strains of mice and hamsters. On the basis of present evidence, which includes the observation that these attenuated organisms are capable of producing progressive disease in the patients

from whom they are obtained, we are in no position to label these organisms as innocent or non-pathogenic. Such patients, who harbor these organisms and discharge them in their sputa, are to be considered as active cases of tuberculosis.

SUMMARY

From the above, it should be apparent that the problem of pulmonary tuberculosis in our midst is far from solved. The physician who searches for, and remains alerted to the presence of pulmonary tuberculosis in his patients will find this disease more frequently than he anticipated. The treatment of the active case requires utilization of all the methods of therapy, which have proved successful in the past and present. The intelligent use of present day antimicrobial agents requires some understanding of their effects on the tuberculous infiltrate as well as their effect on the other systems of the patient's body. Modern bacteriologic technique must be employed in order to have a more careful evaluation of the status of the pulmonary lesion. This requires concentrates, cultures, animal inoculations and sensitivity studies done prior to induction of antimicrobial therapy as well as repeated studies through the course of treatment. Antimicrobial treatment should consist of combined uninterrupted therapy for a minimum of twelve months and for a period of 18 to 36 months depending upon the extent and the nature of the disease in most cases. Expert judgment must be employed to determine whether surgical intervention is necessary, when it is best performed and what the proper surgical procedure should be in each case. Continued medical surveillance of the previously treated case and the newly found inactive case must be continued indefinitely to prevent reactivation and to institute treatment at the earliest time of reactivation when it does occur.

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GENERAL PRINCIPALS OF PHYSICAL DIAGNOSIS OF THE CHEST

HARRY J. ISAACS, M.D.*

Introduction

The art of percussion and auscultation of the chest is, indeed, a difficult one. It takes the examiner many years to achieve perfection in these procedures. I have seen seasoned clinicians, who percuss very awkwardly and imperfectly. In a case of this type, one feels that the clinician is merely "going through the act", and receives little or no information. In fact, one can state that he is more confused from his percussion, than if he had not percussed the patient at all. It is unfortunate, that many physicians rely entirely on the x-ray findings and place little or no value on their own physical findings. It is always advisable to obtain all the information possible by the simple procedures of percussion and auscultation, and then to confirm the findings by a roentgenogram of the chest. It is deplorable that the art of physical diagnosis of the chest is becoming a lost one, and that clinicians are now relying entirely on the laboratory for a clinical conclusion.

One knows that most diagnoses are made by obtaining a thorough history together with a reliable physical examination, and that the various laboratory aids should only be used to confirm the clinical impression. However, it seems, at present, that the emphasis on laboratory work is exaggerated and that the clinical sense and judgment of the examiner are put aside for various laboratory procedures. This is a deplorable state of affairs. The good clinician is characterized by his ability to evaluate the important points of the history, together with a complete physical examination.

Diagnostic Aids

Percussion and auscultation of the chest should be simple and systematic. One may start at the base and percuss to the respective apex, percussing each side of the chest as a unit. He then may make comparisons with the opposite side. Many chests are percussed in a haphazard manner, because the examiner passes from one side of the chest to the other. He may conclude that a respective apex shows dullness, and that the opposite apex shows normal resonance. This may be entirely incorrect, for although one apex may appear dull, and the other, impairment of resonance. If he had percussed each side of the chest as a unit, he would be able to elicit these differences.

The apices should always be percussed as to size (Kronig's isthmus). Changes in the size of Kronig's isthmus are comparatively simple to percuss, and a good deal of information may be obtained. An enlarged isthmus may be due to a spontaneous pneumothorax or a marked emphysema. A contracted isthmus can occur in apical pleural thickening or in an old pulmonary tuberculosis.

Direct percussion of the clavicles may be of value. Marked dullness can occur with a chronic fibroid phthisis. Hyperresonance can occur with a spontaneous pneumothorax or a severe emphysema.

Percussion of the upper axillary regions is very important. Marked dullness, on one side, is indicative of a descending process with marked lung involvement. Resonance, when found over this area, shows that the process is not too extensive. I have seen many cases which show bilateral extensive dullness, with findings presenting themselves at the same level (posteriorly). However, one axilla showed dullness and the other normal resonance. The dull axilla shows a more extensive involvement with a descending process.

Under normal conditions both the upper and lower sternum are resonant. Dullness in the upper sternum signifies a markedly dilated aorta; a substernal

*Professor and Chairman, Department of Medicine, The Chicago Medical School; Attending, Mount Sinai Hospital; Attending, Michael Reese Hospital, Chicago, Illinois.

thyroid; an aortic aneurysm; enlarged lymph glands, due to Hodgkin's disease, leukemia, lymphosarcamoa, infectious mononucleosis or tuberculosis; and dullness can also occur with a large esophageal diverticula. Dullness in the lower sternum may be due to pericardial effusion, congestive heart disease with a congested liver, or an inferior mediastinal syndrome due to a malignant process.

Methods

There are four types of percussion. These are: 1.) heavy percussion; 2.) light percussion; 3.) jumping percussion; and 4.) sliding percussion. Each type of percussion has its distinct value. At times, various types of percussion have to be instituted to obtain the necessary information.

Heavy Percussion

This has a role in the diagnosis of central lesions of the lung. A central pneumonia or a central neoplastic process will only show abnormal percussion findings by heavy percussion.

Light Percussion

This can be used with peripheral lesions of the lung. It is of no value in central pulmonary processes.

Jumping Percussion

This very simple procedure demonstrates the heart borders and the position of the diaphragm. Sudden changes in lung resonance are easily obtained and definite anatomical location of abnormal findings are easily demonstrated. In jumping percussion, one relies on the sense of resistance offered to the percussion fingers. One can be deaf and still use jumping percussion to obtain the necessary abnormal findings of the lung.

Sliding Percussion

This is a light percussion, which presents slight resistance to the findings and is inaudible. It has a definite role in the percussion of the posterior mediastinum. Here, we can conclude that there are enlarged glands in this area: this is found in Hodgkin's disease, tuberculosis, leukemia, lymphosarcoma, infectious mononucleosis or a simple inflammatory adenitis. I, personally, advise all medical students to practice these various types of percussion and to perfect their technique, to the best of their ability. I have seen cases, in which there is dullness to flatness on percussion, which may be quite confusing to the examiner. He is uncertain as to whether these findings signify fluid, consolidation or a thickened pleura. A pleural effusion will show a change at a higher level with light percussion, and a change at a lower level with heavy percussion. A markedly thickened pleura will show a change at the same level with both light and heavy percussion. The same is true for a massive lung consolidation.

Extrapulmonary Compression

Percussion of a normal apex shows normal resonance. When dullness or impairment is present, it is usually indicative of intrapulmonary apical pathology. Occasionally, apical dullness may be due to extrapulmonary compression, *i.e.*, cervical rib, adenoma of the thyroid, or cervical adenopathy. The clinician, is then at a loss to explain these abnormal percussion findings. As a result, many patients have been labeled as having pulmonary tuberculosis due to the presence of dullness and rales. A very simple bedside procedure can differentiate intrapulmonary apical pathology from extrapulmonary apical compression. A normal resonating apex shows an increased resonance on bending the patient forward. If extrapulmonary apical compression is suspected, bending the patient downward causes a disappearance of the dullness and rales previously found on percussion. A typical example of pulmonary compression is found in pericardial effusion with pneumonia-like findings located posteriorly at the angle of the left scapula. This is called Bamberger's or Ewart's sign. Bending the patient forward, or placing the patient in the knee-chest position, will cause the disappearance of all the abnormal physical findings. A good dictum to follow is this: whenever a young patient has a pneumonic process situated at the angle of the left scapula posteriorly, be sure that you are not dealing with a pericardial effusion with compression atelectasis of the left lung.

Brown Induration of the Lung

A very confusing physical finding is that of dullness and rales at one or both apices in a patient who has a definite mitral stenosis. Mitral stenosis can cause brown induration of the lungs affecting either one or both apices. This is quite often diagnosed as pulmonary tuberculosis. One knows that pulmonary tuberculosis and mitral stenosis are very antagonistic, and that apical lung findings in a definite case of mitral stenosis are seldom due to a superimposed pulmonary tuberculosis. Only if a positive sputum or cavitation is found, can the diagnosis of a superimposed pulmonary tuberculosis be added. I have seen many massive tuberculous pleural effusions diagnosed as pneumonia. With exudates, such as those found with tuberculous pleural effusion and post-pneumonic empyema, one can find tubular breathing and rales, together with increased tactile and vocal fremitus. However, when flatness is present on percussion, irrespective of what other findings are present in the lung, always think first of a pleural effusion.

Lobes of the Lung

For practical purposes, percussion of the anterior right chest demonstrates the upper and middle lobes; of the left chest, only the upper. Posteriorly, percussion reveals on the right side, only the upper and lower lobes; on the left, both upper and lower lobes. The right middle lobe is best percussed and auscultated in the mid-axilla and the lower portion of the right chest anteriorly.

Posterior Mediastinum, Spleen and Retroperitoneal Regions

I advise students to perfect their percussion techniques of the posterior mediastinum, the spleen and the retroperitoneal regions. I admit that retroperitoneal percussion is not as accurate as that of the lung. However, there have been many times when percussion of that area would speak for some retroperitoneal pathology, and that further work-up may show evidence of retroperitoneal Hodgkin's disease, sarcoma, or hypernephroma of the kidney. Definite dullness would be obtained in cases of retroperitoneal pathology.

Auscultation

Auscultation, as an art, seems to be mastered more easily than percussion. Many students rely on their auscultatory findings, and fit their percussion findings to suit the abnormal sounds heard. I, personally, advise students to percuss the chest first, and to master this art by bedside experiences. Any rale, to be of any significance, must be constant. On auscultation of the first few inspiratory phases, a few rales can be heard at the bases, only to disappear later. These rales are usually due to a partial atelectasis and are inconsequential. There are two types of rales that disappear in a day or so; those due to either atelectasis or to congestive heart failure from passive congestion of the lungs.

Persistent apical rales can occur with a pneumonic, tuberculous or bronchiectatic process. In fact, the only physical signs of activity of a tuberculous process are those of persistent rales and amphoric breathing. It is a well known fact, that fifty or more per cent of all cavities are silent. This is due to the fact that the cavities are either too small, are filled with secretion, or do not communicate with a bronchus. Pericardial and pleuritic friction rubs are sometimes difficult to hear. They are usually not constant. A pericardial rub may be intensified by sitting or leaning the patient forward and applying pressure with the stethoscope.

A very interesting phenomenon to observe, is the relationship of the duration of the disease with the number of rales heard. If the patient is sick for only a day or so, and on auscultation many crepitant rales are heard, then one can state, that the multitude of rales heard play no relation to the present condition. One can assume these rales have been there for many months, and that the underlying pathology may be that of a chronic bronchiectasis or pulmonary tuberculosis.

I am forced to admit that x-rays of the chest give the clinician a great deal of information and, at times, they may be the deciding factor in the diagnosis of

the underlying pathology. Portable x-rays of the chest may be very atypical and bizarre, and lead one to many diagnostic errors. There are many chest diseases, such as, tuberculosis, sarcoidosis, miliary tuberculosis, lung infarcts, *etc.*, in which the physical findings are either nil or else very insignificant, while the x-ray shows extensive pathology. At times, I have wondered myself whether I had mastered the art of physical diagnosis of the chest, especially where there is a marked disproportion between negative physical findings and very definite evidences of demonstrable roentgenographic pathology. However, although these differences do occur, the student should not be discouraged to further his experiences in the art of percussion and auscultation. Only after years of experience will he have mastered this art, and he can then, be classified as an "excellent clinician". These qualifications sometimes spell the difference between success and failure in the practice of medicine.

Physical Diagnosis of Diseases of the Chest

Many diseases of the chest may present the same physical findings. The final interpretation and diagnosis may then rest with the bacteriological workup, the progress of the disease, or an exploratory thoracotomy with a definite lung biopsy. At times, a diagnostic needle biopsy of the lung may be pathognomonic. Personally, due to the dangers involved, I do not recommend this procedure. A collapsed lung or a massive hemorrhage may result from a needle biopsy. Many extensive cases of pulmonary tuberculosis may show little or no findings. In the absence of lung findings, one should be quite aware of certain disease processes, *i.e.*, incipent pulmonary tuberculosis, solitary "isolated" lesions (coin) of the lung and extensive metastasis to the lung.

In conclusion I plead for a better understanding of the art of percussion and auscultation. Only after years of clinical experience, can one master this art.

INJURIES TO THE CHEST

SAUL A. MACKLER, M.D.*

A comprehensive consideration of chest injuries necessitates the adoption of a classification not only for the purpose of completeness, but as a basis for understanding the interrelationship of various wounds and concomitant intrathoracic damage. The accompanying chart outlines the possible types of injuries which may be incurred by the chest, and the intrathoracic complications which may be their counterpart.

Injuries of the chest may be divided, in the classical manner, into two gross categories; namely, penetrating and nonpenetrating (blunt) wounds. Of equal importance to an identification of the specific type of wound, is the recognition of the presence of concomitant intrathoracic damage as a complicating factor. It is to be noted that any injury of the thorax, whether penetrating or blunt in nature, may give rise to any one or a combination of the listed intrathoracic complications. To be sure, certain forms of internal complications are more prone to accompany particular wounds, but it must be stressed that any complication may result from any form of injury sustained by the thorax.

Because of the limitation of space, items from the accompanying classification will be chosen for discussion according to the frequency of their appearance in the ordinary accidents of civilian life. Less common injuries, such as thoracoabdominal wounds, will of necessity be omitted from this presentation.

Intrathoracic Damage

It is a common fault to overlook the existence of intrathoracic complications following a moderately severe injury to the chest. It is usual to find that a multiplicity of roentgen films have been obtained in various positions and exposures in an attempt to demonstrate the existence of a rib fracture. Yet, no simple upright film was ordered to determine the status of the intrathoracic viscera. While the former may be necessary for legal reasons, the latter is necessary for medical purposes.

PNEUMOTHORAX

The most common intrathoracic complication is that of pneumothorax. The accumulation of air in the pleural cavity, occupying space usually taken by the lung, is due, paradoxically, to the lung itself. The escape of air from the lung is readily comprehended following a penetrating wound in which the lung itself is pierced: it is less readily appreciated following blunt injury.

The pneumothorax that appears as a consequence of a blunt injury results in most instances from a laceration of the lung caused by the raw end of a fractured rib. It may be difficult to conceive how a fractured rib may penetrate the lung when the roentgenogram shows no displacement of the opposing margins of the rib at the fracture site. Indeed, it is sometimes most difficult to discern the region of the break itself, no less to visualize a lacerating edge. It must be realized that the rib, because of its quality of resiliency, will snap back to a position of alignment immediately after the force causing the fracture is removed. The separation of the fragments, then, exists only during that fraction of time when the blow is applied to the chest, and it is at this instant that one end momentarily invades the chest cavity. The distance that is traversed to reach the lung is not great; it is measured in micra. The visceral surface of the lung lies in direct apposition to the parietal pleura, which in turn is in contact with the inner surface of the rib periosteum.

A pneumothorax is a frequent companion to blunt trauma in children and young adolescents, yet rib fractures are rarely seen at this age. Fracture is rare because of the malleability of the rib, its abutment in cartilage anteriorly, and the extreme mobility of the chest cage as a

*Assistant Professor, Department of Thoracic Surgery, The Chicago Medical School.

INJURIES OF THE CHEST

Non-Penetrating (Blunt)	*Concomitant Intrathoracic Damage (Complications)*
Fracture of rib	Pneumothorax Tension pneumothorax
Flail chest	Hemothorax Continued intrapleural bleeding
Costo-chondral disarticulation	Emphysema a. Subcutaneous b. Mediastinal
Fracture of sternum	Air channel obstruction a. Atelectasis b. Edema (wet lung)
Traumatic cyanotic asphyxia	Cardiac injury a. Myocardial contusion (blunt) b. Tamponade (penetrating)
Blast injury	Diaphragmatic injury a. Rupture (hernia) (blunt) b. Perforation (thoracoabdominal) (penetrating)
Penetrating	Esophageal injury a. Rupture (blunt) b. Perforation (penetrating)
Wounds not requiring thoracotomy	
Wounds requiring thoracotomy (indications) 1. Diaphragmatic perforation (thracoabdominal) 2. Retained missiles 3. Mediastinum — missiles traversing or lodging within 4. Bronchial wounds 5. Continued intrapleural bleeding	

whole. How then is the lung injured to permit the escape of air to form a pneumothorax? The sudden application of a blow to a mobile chest cage effects a momentary forceful compression of the underlying lung with a concurrent, precipitous rise in the intrapulmonic pressure. This abrupt rise may cause the rupture of interalveolar septae, and if the tear occurs toward the visceral surface, air will escape into the pleural space. The mechanism is akin to the bursting of a balloon when stepped upon.

Little need be said regarding the diagnosis of a pneumothorax. The history of trauma, a tympanitic percussion note, and the absence of breath sounds are classical findings. A simple, upright roentgen film should be taken in all patients with trauma to the chest. The presence of a pneumothorax will then be found although physical findings may be lacking.

Treatment

Treatment consists of the application of the elementary principle governing the management of all forms of thoracic trauma. Simply stated, this asserts that anatomic structures shall be made to resume their normal configuration and spatial relationships. Since air now usurps the space previously occupied by the lung, it must be evacuated from the pleural cavity so that the lung may once again resume its normal position in a state of expansion. This air may be removed manually by thoracentesis, or may be permitted to decompress itself by means of an indwelling needle or catheter and a water seal system. Evacuation of the pneumothorax should be begun within the first twenty-four hours. Greater promptness is urged by the presence of dyspnea. The quantity of air to be removed at this time is dictated by the subjective appearance of a sense of constriction or tightness within the chest. This is a manifestation of the development of an increasingly high negative pressure within the pleural space as a result of the inability of the lung to expand rapidly enough to keep pace with the rate of air withdrawal. At this point, thoracentesis should cease, to be resumed within the next twenty-four hour period. It may be necessary to perform two to four thorcenteses, at twelve to twenty-four hour intervals, to empty the pleural space. The progress of the lung should be followed by fluoroscopy or roentgenograms.

Objection is frequently voiced at this manner of treatment and the recommendation is made for conservatism without direct mechanical interference. Proponents postulate that the free air will ultimately be absorbed by the tissues. That such air will eventually be absorbed, in

most cases, in a matter of ten days or two weeks depending upon the quantity present, is undeniable; yet to allow the body to slowly rehabilitate itself in this fashion is fraught with the risk of the lung remaining permanently unexpandable. In view of the trauma, there is invariably present some quantity of free blood in the pleural space which is in a state of constant motion; not only by diaphragmatic and cardiac action, but by the bodily movements of the patient, upright or reclining, prone or supine. During these activities, the free blood flows over the surface of the lung and there is a slow but constant precipitation of fibrin upon its visceral surface. Eventually, a sufficient deposit will have been made to effectively coat the lung and prevent its re-expansion even if active removal of the pneumothorax is now begun. A thoracotomy with decortication of the lung will then be necessary to release it from this encasing film.

HEMOTHORAX

The blood that accumulates within the pleural space has its origin in two possible sources; namely, the systemic circulation of the parietes (intercostal or internal mammary vessels) or the pulmonary circulation. Because of its voluminous size and structure, the lung is most frequently involved and it is the usual source of bleeding.

The diagnosis presents little difficulty. The history of trauma and the physical findings of dullness to percussion and absence or diminution of breath sounds indicates a hemothorax. Because the lung is so frequently the site of injury, air as well as blood will have escaped, (hemopneumothorax), and a roentgenogram taken in an upright position will demonstrate the blood-air level. This will not be demonstrable if the film is obtained with the patient in a supine or prone position.

Treatment

In treatment, the basic principle is once again enforced; namely, evacuation of the pleural space so as to permit the lung to resume its normal anatomic position in a state of expansion.

The regime of emptying the thoracic space by daily thoracentesis is as described in the treatment of pneumothorax. At the time of the initial thoracentesis, if less than twenty-four hours has elapsed from the time of injury, the evacuated blood may be returned to the circulation as an autotransfusion. Instead of discarding the blood, a length of plasma tubing leading from the stopcock-thoracentesis system is connected to a sterile flask containing citrate solution. Commercially prepared transfusion bottles containing sufficient citrate solution to prevent the coagulation of the usual 500 cc. of blood drawn from donors are easily available and lend themselves most conveniently for the collection of the intrapleural blood. Typing and cross matching is unnecessary, and the blood may be infused immediately into the venous circulation. Only two conditions restrict the utilization of autotransfusion: the presence of a thoraco-abdominal wound in which one suspects that intestinal or gastric content may have been aspirated into the chest via the diaphragmatic wound, and the age of the hemothorax. If the latter has existed for a period exceeding approximately twenty-four hours, autotransfusion should not be done because of the danger of introducing free hemoglobin into the circulation with the possibility of nephrotic consequences. Stagnation and the constant motion imparted to the pooled blood by the unceasing activity of the diaphragm, heart and chest wall contributes to corpuscular fragmentation and the release of free hemoglobin.

The same voices raised in objection to mechanical intervention in evacuating a pneumothorax will be heard concerning like treatment for hemothorax. Similarly, but more emphaticaly, recalcitrance in removing free blood from the pleural space, in anticipation of ultimate absorption by the tissues, will result in incarceration of the lung and/or loculation of the hemothorax by the precipitation of fibrinous septae throughout the collection. To empty the pleural cavity by thoracentesis or by catheter and water-seal is now impossible, and a thoracotomy indicated. Further, reluctance

to empty the thoracic space invites the development of an empyema, antibiotics notwithstanding. The ideal conditions for bacterial growth are present: a warm, moist, dark atmosphere—an excellent culture medium, and contamination from the bronchial tree or, in a penetrating wound, from the outside.

Continued Bleeding

To the initiate in the treatment of thoracic injuries, the easy withdrawal of several hundred cubic centimeters of blood from the chest may induce a sense of panic prompted by the thought that instead of treating, one is exsanguinating the patient. It is to be realized that the blood which is withdrawn is blood that has accumulated, and that has been carried by the pleural cavity as a natural receptacle from the time of trauma. It is not unusual to collect as much as 1500 cc. to 1800 cc. of blood in the pleural space before the source becomes controlled. One need not be concerned that a large vessel is involved. Had such been the case, the patient would have long since expired due to hemorrhage. To be sure, continued intrapleural bleeding does occur, but with great infrequency. This possibility does not contraindicate thoracentesis upon the assumption that evacuation will stimulate bleeding by removing the collection as an "intrapleural tampon". There is no indication to suggest that accumulated blood will act in such a hemostatic manner. Before sufficient pressure could be brought to bear to stem even the flow of the pulmonary circuit, which has a pressure one-fourth that of the systemic circulation, the patient would have expired of asphyxia due to compression. Indeed, clinical experience shows that the converse is true. By emptying the pleural cavity and expanding the lung, the apposition of tissue surfaces will tend to seal the lacerated areas.

The suspicion of continued intrapleural bleeding, thus, does not contraindicate aspirations of the pleural cavity, but indeed demands such. It is by this means that the diagnosis is ascertained. In the treatment of the usual hemothorax, it is the adopted procedure to voluntarily remove only 500 cc. of blood at the first thoracentesis. This is an arbitrary quantity, but is selected because it is adequate to give increased pulmonary volume, and is the amount given as an autotransfusion, assuming that the injury is of recent origin. Furthermore, prolonged manipulation is tiring not only to the patent, but to the surgeon as well, and the existence of pain and circulatory instability is not conducive to an ideal state of patient cooperation. The succeeding aspiration is performed upon the following day, at which time complete evacuation is attempted. A third, or a fourth trial may be necessary on successive days if a roentgen film shows a respectable residue.

The suspicion of continued intrapleural bleeding, however, alters the time-table of daily aspirations. This possibility is to be considered if the circulatory status fails to improve as anticipated following adequate blood replacement, and the administration of other supportive measures. A second thoracentesis is performed at this time instead of waiting for the following day, and the pleural cavity is emptied of its contents as completely as possible. At the expiration of an additional three to four hours, thoracentesis is repeated, particularly if circulatory stabilization has not been attained despite continued treatment. If at this time blood is easily obtained again, and the total quantity of the three aspirations exceeds 1800 cc. to 2000 cc., a source of continued bleeding must be assumed. If doubt persists, delay for an additional interval, followed by thoracentesis, may be necessary to arrive at a conclusion.

That continued intrathoracic bleeding is an uncommon occurrence deserves reemphasis. Hence the precaution of establishing the presence of the condition by the method of repeated thoracenteses and measurement of the quantity of blood withdrawn. If such measures were not taken, many unnecessary thoracic explorations would be undertaken upon precipitous judgment, hastened by the appearance of a large quantity of blood obtained by a single thoracentesis.

Method of Aspiration

Aspiration of the pleural space is so common a maneuver in the treatment and diagnosis of chest injuries, that the simplification of the materials and method would be of great advantage. A simple thoracentesis system consists of a 50 cc. syringe and a 15 gauge, short bevel needle. A three-way stop-cock, which may be obtained from a spinal manometer set, is interposed between syringe and needle to eliminate the awkward necessity of detaching and reattaching the syringe and needle to discard the aspirated material. A short length of plasma tubing may be fixed to the side vent of the stop-cock for neatness in the manner of disposing, (or collecting for auto-transfusion), the evacuated contents. The manner in which the lever of the stop-cock controls the flow should be ascertained prior to the thoracentesis, because in dealing with a medium which is invisible, as in a pneumothorax, the mistake may be made of introducing air into the chest rather than removing it. The site chosen for the insertion of the needle should be such as to allow the patient (and surgeon), a position of comfort throughout the period of the procedure. The usual location upon the posterior thoracic wall at the angle of the scapula is not only mutually discomforting, but compels the patient to adopt a sitting position, which he might not be able to assume because of associated injuries, pain, coma or shock. A site which is easily accessible, and allows freedom of movement, is the mid-axillary region at the transverse level of the nipple. The intercostal spaces are easily palpable here, being free of heavy, over-lying musculature, and the area is made readily available by slightly abducting the elbow. Thoracentesis is thus accomplished without discomfort to the patient by any change in the usual reclining or semi-reclining position. An alternate site is the second intercostal space anteriorly, at the mid-clavicular line. This should be selected, however, only for the evacuation of a pneumothorax, as it is air, rather than fluid, which will collect within the upper region of the hemithoracic space.

SUBCUTANEOUS EMPHYSEMA

This condition is noted for its marked effect upon the visitor rather than upon the patient. The accumulation of large quantities of air in the subcutaneous tissues, particularly in lax regions as the scrotum, neck, and eye-lids, produces no ill effects other than that of moderate discomfort, but the distortion may be so grotesque as to make one unrecognizable. The feeling of crepitus upon palpation distinguishes subcutaneous emphysema from edema.

Treatment

The treatment of subcutaneous emphysema is directed toward its origin: to prevent further accumulation, rather than toward the condition itself. Once the source of supply of air is controlled, the subcutaneous space eventually decompresses itself by the process of tissue absorption. This may take one week to ten days depending upon the extent of emphysema. Skin incisions with undercutting for the purpose of decompression are not to be condoned, as they serve no purpose except to open avenues for infection.

The source of air is usually an accompanying pneumothorax. During cough, or other exerted movements, there is a marked rise in intrathoracic pressure, as a result of thoracic space compression. Each momentary compression results in increasing the pressure of the free air within the pleural cavity, and a vent will be sought for its escape. In blunt injuries, a rib fracture is usually responsible for the development of the pneumothorax and this site, with its accompanying parietal pleural rent, serving as a means of exit. Each cough or exertive movement forces a small quantity of air into the soft tissues of the chest wall.

Treatment concerns itself with managing the pneumothorax. Occasionally, the expected pneumothorax responsible for the subcutaneous emphysema is not found upon roentgen examination. In such instances, the absence of a pneumothorax is accountable in two ways: a pneumothorax previously was present, but had completely evacuated itself into the subcutaneous tissues in the manner

described, or a free pleural cavity might not have existed prior to the injury in which air could collect. A former pneumonitis with pluritis might have obliterated the potential pleural space by the formation of visceral-parietal pleural synechia. In such instances, where there is surface to surface adherence of lung to inner thoracic wall, there is a direct communication of wounds, and air is then expelled directly into the soft tissues of the chest wall. Excessive degrees of emphysema are found in such cases. Continued inflation of the subcutaneous spaces may be prevented by the application of a firm adhesive dressing over the site of injury. Palpation of the point of tenderness discloses the region of fracture in blunt injuries, and local compression of the overlying loose muscle and subcutaneous tissue prevents the further escape of air.

MEDIASTINAL EMPHYSEMA

The means whereby this form of emphysema develops is sometimes obscure. Like the subcutaneous variety, it owes its origin, in many instances, to an underlying pneumothorax. Just as air is forced from a pneumothorax into the soft tissue spaces of the chest wall by repeated exertions, in a like manner, air may find its way to the mediastinal pleura. It is possible that the tug of a collapsing lung, adherent at its mediastinal surface, may contribute to such a tear.

A less direct route to the mediastinum is also available by way of arterio-bronchiole pathways. Although a deep laceration of the lung will be followed by the escape of air into the pleural cavity, air may also gather within the depths of the wound. Such a collection in the substance of the lung, yet not contained within the normal alveolar unit, is a *pneumatocele*. Once again, repeated acts causing momentary forceful rises in intrathoracic and intrapulmonic pressure not only aid in contributing to the collection, but force it along in search of a pathway of low resistance. The loose, areolar tissue of the arterio-bronchiole sheath is such a pathway. Air is thus intermittently propelled along the arterio-bronchiole path, from small to larger radicals, until the main channels at the hilum, where it eventually dissects directly into the mediastinum.

Mediastinal emphysema may be found without the presence of a coexisting pneumothorax. Its absence is attributed to the same mechanisms responsible for its occasional absence in subcutaneous emphysema; namely, loss of the pneumothorax because of its complete expulsion into the mediastinal space, or because of pre-existing obliteration of the pleural cavity by visceral-parietal synechia. In the latter situation, a pneumatocele formed within the depths of the lung wound will dissect to the mediastinum via the arterio-bronchiole pathway, as described. A pneumatocele may also be formed following a blunt injury, without direct laceration of the lung. A blow upon the chest, particularly in children and adolescents, may result in the disruption of alveolar septae without the fracture of a rib. As previously described, if this occurs at the surface of the lung, a pneumothorax will form. If, however, the disruption occurs within the lung substance, without involvement of its visceral surface, a pneumatocele will be produced which will contribute to a mediastinal emphysema, but without the intermediate formation of a pneumothorax.

The diagnosis of mediastinal emphysema is to be suspected by the appearance of a subcutaneous emphysema at the base of the neck, extending upward toward the jaw and face. Air within the mediastinal space, as it continues to accumulate, ascends to the superior thoracic strait through which it passes, to appear within the tissue spaces of the neck. This is the route of least resistance, because the diaphragm bars any progression in a caudal direction, whereas the tissue spaces of the mediastinum are directly continuous with those of the neck through the superior aperture of the thoracic cage. Auscultation of the precordium may aid the diagnosis if one elicits the sounds of crepitation synchronous with the heart beat.

Treatment

The treatment of mediastinal emphysema, like subcutaneous emphysema, resolves itself in the management of the underlying cause. Direct surgical intervention to decompress the mediastinum by means of a cervical incision is almost never necessary. Of all the tubular anatomic structures harbored within the mediastinum, the tracheo-broncial tree, as designed by nature, is most capable of resisting compression: the venous system among the least. Hence, when one is forced to contemplate surgical decompression because of unrelieved dyspnea, one must be assured that there is present sufficient compression of the mediastinal structures to at least impede venous drainage. This may be determined grossly by inspection of the external jugular veins of the neck. However, in almost all instances, the dyspnea can be attributed to causes other than emphysema of the mediastinum.

The escape of air directly into the mediastinal (and/or pleural) space from a wound of a large bronchus is a rare occurrence. When encountered, it is likely to follow blunt injury associated with massive trauma to the thorax and body. The brunt of sudden torsion and weight of the lung mass is borne by the large bronchi at points of fixation at the hilum. Fracture, tear, or complete separation of the bronchus or trachea may be the direct result. Similar wounds of the major bronchi, consequent to penetrating missiles or weapons, are not usually compatable with life because of concomitant injury to the contiguous blood vessels. Death rapidly follows, either from hemorrhage or from asphyxia due to flooding of the bronchial tree.

It must be mentioned that mediastinal emphysema, subcutaneous emphysema, and pneumothorax may be produced by means other than that described. In rare instances, these body spaces may be invaded by gas from an alimentary rather than a respiratory source. For example, rupture of the esophagus will produce mediastinal emphysema, and eventually pneumothorax. Free intraperitoneal air or gas resulting from a perforation of a hollow viscus may ascend via the diaphragmatic hiatus into the mediastinum. Injury to the retroperitoneal portion of the duodenum will permit the dissection of air into the retro-peritoneal space, and thence into the subcutaneous tissues of the flank, abdominal wall and scrotum.

ATELECTASIS

The accumulation of material within the bronchial channels is to be regarded as an expected accompaniment of chest injuries rather than as a complication. An inattentive attitude regarding the maintenance of clear passageways will permit such an accumulation, with edema and atelectasis as a consequence. Blood and secretions will collect within the bronchial tree because, following injury, the usual activity of the respiratory tract is obtunded and adequate respiratory excursions and cough are prevented by pain. The chest cage is voluntarily held immobile or splinted, and pulmonary exchange is accomplished primarily by diaphragmatic activity. Because of this imposed limitation upon the depth of respiratory movement, an increase in the respiratory rate compensates to effect an adequate exchange of gases. The dyspneic patient, then, frequently owes his deficient respiratory status to thoracic wall pain rather than to an encroachment upon his vital capacity by an intrathoracic complication.

Treatment

Patency of the bronchial passageways is best maintained by the natural mechanisms of adequate depth of respiration and the cough reflex. These will be, respectively, voluntarily and involuntarily restricted by the pain which is elicited by motion of the thoracic cage. Alleviation of pain, therefore, is of concern not only as a palliative measure, but as an active form of treatment. Narcotic drugs may be used, but must be limited to avoid depression of the central nervous system, which will also contribute to immobility. The method of choice is the local relief of pain by means of an intercostal nerve block. The inferior edge of each rib to be anesthetized is selected and 2.0 cc. of a one-half per cent solution of procaine hydrochloride is injected into the intercostal space. It is necessary

to block several interspaces above and below the rib or ribs which are injured. The usual site is the posterior aspect of the thorax, approximately three inches from the midline, so that a physiologic interruption of nerve impulses is accomplished between the site of injury and the nerve root. The precaution of drawing back upon the plunger of the syringe prior to injection should always be taken as the intercostal vessels course along the inferior margin of the ribs, and they may be pierced.

Once the pain has been relieved, the patient is to be actively encouraged to take deep respirations and to cough. A "cough regime" is enforced in which the patient is required to cough for three to five minutes at half hour intervals. If the patient is uncooperative, the cough reflex may be stimulated by inserting a nasal catheter to the region of the epiglottis. Because of the unpleasantness of this method, the sight of the catheter alone may soon condition a cough reflex.

Inability to clear the bronchial passages by natural means, because of associated injuries, coma or shock, will require active measures on the part of the surgeon to obtain and maintain patency. The most effective method is by bronchoscopic aspiration. However, lacking properly trained personnel or equipment for this procedure, effective use may frequently be made of catheter aspiration of the tracheobronchial tree.

Catheter Aspiration

Catheter aspiration often is mentioned glibly, but its actual execution is usually thwarted, most frustratingly, by the natural mechanisms which guard the laryngeal additus against the entry of any foreign substance. The following method is one in which insertion of the catheter will be successfully accomplished most frequently. First, the posterior pharynx and base of the tongue is lightly sprayed with a 2.0% solution of pontocaine hydrochloride to obtund the cough and gag reflex. The tongue is then firmly pulled from the mouth using a gauze square to prevent its slipping. This maneuver moves the epiglottis forward and helps to bare the glottis. A nasopharyngeal tube is now inserted via one of the nares, and is held so that the tip remains poised in the region of the posterior pharynx. The patient is then requested to cough voluntarily, and at the moment that expulsion of air starts, the tube is quickly thrust down. The vocal cords are widely separated at this moment to permit the sudden release of the increased intratracheal pressure, and it is at this instant that introduction of the catheter through the unwary glottis may be most successful. A number 16 Fr. Levine tube is preferable to the ordinary catheter, as the latter has insufficient length to reach the carina or beyond, to cleanse the main bronchi.

SUMMARY

A classification of chest injuries is presented, outlining the types of wounds which may be sustained by the thorax. Of equal, or greater importance to the identification of the variety of wound, is the recognition of the existence of concomitant intrathoracic damage and the universality of its appearance following any form of trauma. These complications are similarly listed, and the most common discussed from the standpoint of pathologic physiology, diagnosis and treatment.

SOME CLINICAL ASPECTS OF PULMONARY FUNGUS DISEASES

GERSCHEN L. SCHAEFER, M.D.*

The frequency with which pulmonary fungus diseases are being diagnosed has removed them from the realm of the rare or unusual. With increased ease in travel and since so many members of our armed forces have been stationed in endemic areas, a greater percent of our population have been exposed to exogenous fungi. Prolific use of antibiotics[1-8] and corticotrophins[9,10] has enhanced the growth and pathogenicity of endogenous fungi. These facts, coupled with better diagnostic laboratory procedures, excellent public health reporting of cases, and the location of previously unrecognized endemic areas have obliged physicians to become better acquainted with this group of diseases.

Table I enumerates the more common fungi found in the United States that may invade pulmonary tissue together with their source, other organs they invade, and therapy used. A brief general discussion of the group will follow, and a somewhat more detailed description of the more common exogenous fungi: North American Blastomycosis, Coccidioidomycosis and Histoplasmosis.

Symptoms

The symptoms of all pulmonary fungus diseases are dependent upon the pathologic process present, the extent of involvement, the resultant encroachment on pulmonary function, and the systemic reaction to a pathogenic invader. The pathology produced is not specific. The specific organism per se, therefore, is not responsible for the initiation of symptomatology. It is for this reason that the symptoms of fungus infection are not specific. It may resemble any bacterial, viral, or neoplastic process, depending upon the form it takes, the degree of involvement and individual response to the presence of the fungus, which varies from patient to patient.

Initial symptoms are frequently catarrhal or pneumonic. Hemoptysis is not uncommon in blastomycosis[11,12], coccidioidomycosis[13], candidiasis, geotrichosis, aspergillosis, actinomycosis[14], and occurs occasionally in cryptococcosis and histoplasmosis[15]. Wheeze may be present if there is bronchial involvement. Those symptoms of mycoses resembling malignancy, such as weight loss, anorexia, and anemia, are not uncommon. Symptoms that are present will indicate only the organ system involved, and not the etiological agent.

X-Ray

The x-ray findings are again non-specific. The picture might resemble pneumonia[11,12,16-18], carcinoma[12,19], lung abscess[12], tuberculosis[19-20], or any granulomatous process with or without miliary involvement[11,21-26]. Cavitation may be found in coccidioidomycosis[13,18,27], histoplasmosis[16,28], blastomycosis[12,15], aspergillosis, geotrichosis, and occasionally in cryptococcosis[29]. Pleural effusion or empyema may be present[15]. Spontaneous pneumothorax has been reported[13].

Diagnosis

Definitive diagnosis can be made only by isolation and identification of the specific fungus. The part of the country in which the patient resides, or has visited, might suggest some of the exogenous fungi. The presence of lesions in other organs might lead one to the diagnosis. However, presumptive diagnosis can be made by skin test, or serologic tests such as complement fixation, agglutination, or precipitin tests in coccidioidomycosis, blastomycosis, histoplasmosis, and sporotrichosis. It must be stressed that isolation of the organism is imperative for absolute diagnosis either by direct examination by experienced personnel, or by cultural methods. Animal innoculation may be useful in cryptococcosis, sporotrichosis, nocardiosis, and histoplasmosis. Mention should be made of the acid fast

*Clinical Assistant, Department of Medicine, The Chicago Medical School; Assistant Director, Chest Department, Mount Sinai Hospital, Chicago, Illinois; Assistant Medical Director, Winfield Hospital, Winfield, Illinois.

TABLE I — PULMONARY FUNGUS DISEASES

DISEASE	FUNGUS	SOURCE	OTHER ORGANS INVOLVED	DIAGNOSIS	TREATMENT
Actinomycosis	*Actinomyces bovis* and other species	*Endogenous* in "normal" mouth	Cervicofacial area, colon, liver, kidney, spine, pleura, intestine	Demonstration of organism—direct and culture	Antibiotics: sulfonamides, penicillin, broad spectrum. 2-OH stilbamidine, KI, Surgery
Aspergillosis	Several species of aspergillus usually *A. fumigatis*	Primarily grain dust	Acces. nasal sinuses, eye, genitals, meninges, spine, ribs	Very common lab. contaminant. Must have *repeated* demonstrations of *branching hyphae* in sputum.	KI
North American Blastomycosis	*Elastomyces dermatidies*	Soil	Skin, bones and joints, liver, spleen, kidneys, prostate, lymph nodes, pericardium, meninges	Demonstration of organism, skin test. Complement fixation.	2-OH stilbamidine, KI, Surgery
Candidiasis (Moniliasis)	*Candida albicans*	*Endogenous* in "normal" skin & mucous membrane	Skin, upper resp. mucosa, vaginal & colonic mucosa, bronchial mucosa, bones and joints, meninges, endocardium	Demonstration of organism, *constantly*, and in *large numbers*	KI, I.V. gentian violet, ethyl iodide inhalation
Coccidioidomycosis	*Coccidiodes immitis*	Soil	Skin, meninges, lymph nodes, bones & joints, testes, abdominal organs	Demonstration of organism. Skin test. Precipitin test. Complement fixation test.	2-OH stilbamidine, (?), prodigiosin, Surgery
Geotrichosis	Several species of *Geotrichum*	*Endogenous* in "normal" mouth and intestinal tract	Oral mucosa, intestines	Demonstration of organism directly and by culture	KI, autogenous vaccine (?)
Histoplasmosis	*Histoplasma capsulatum*	Soil	Reticulo-endothelial system, lungs, liver, spleen, kidney, intestine, oral mucosa, adrenals	Demonstration of organism, animal innoculation, skin test, complement fixation test.	2-OH stilbamidine, ethyl vanillate, Surgery
Mucormycosis	*Mucor*	Soil, manure, fruits	Eye, meninges, brain, intestine	Demonstration of organism	.....
Nocardiosis	*Nocardia asteroides*	Soil	Subcutaneous tissue, brain, meninges, peritoneum, bones	Demonstration of organism, animal innoculation	Sulfonamides
Penicilliosis	*Penicillium*	Dust, soil	Bladder, kidney	? Constant contaminant of air	KI
Sporotrichosis	*Sporotrichum schenckil*	Soil, wood, plants	Lymph glands & ducts, skin, mucous membrane of upper respiratory tract, bones, joints, kidneys, testes, epididymis	Demonstration of organism, animal innoculation, skin test, agglutinations, complement fixation test	KI, Surgery *contra-indicated*
Torulosis (Cryptococcosis)	*Cryptococcus neoformans (Torula histolytica)*	Soil (endogenous-skin?)	Skin, oral mucosa, joints, muscles, bones, spleen, kidney	Demonstration of organism, animal innoculation	Sulfonamides, KI, Surgery

qualities[30,31] of nocardia which on direct smear may be mistaken for tubercle bacilli. Nocardia may survive concentration techniques for the tubercle bacilli even with the use of tri-sodium phosphate and sodium hydroxide as digestants[109]. Observing the organism in surgical or biopsy specimens will be, at times, the only way to make the diagnosis.

Differential Diagnosis

The first disease to be considered in the differential diagnosis of fungus disease is tuberculosis. The similarity of the symptoms, physical findings and x-ray picture makes the diagnosis difficult. There may be spinal involvement and psoas abscess in actinomycosis, aspirgillosis, and blastomycosis. There may be associated meningeal or cerebral involvement in actinomycosis[74,75], cryptococcosis[76-81], nocardiosis[82,83], aspergillosis[84,85], blastomycosis[11,36,86-88], histoplasmosis[89], and mucormycosis. Addison's disease has been reported due to histoplasmosis[28]. To make it more difficult, coccidioidomycosis[90,91], histoplasmosis[92], aspergillosis, candidiasis, sporotrichosis, and geotrichosis may exist concurrently with tuberculosis. *Coccidioides* and the tubercle bacillus have been reported together in the same pulmonary cavity[91]. The fungus diseases may follow a similar course[20] as that of tuberculosis, including the formation of a primary complex in histoplasmosis[16,89], blastomycosis, and coccidioidomycosis[93]. Breakdown of a healed lymph node has been reported in coccidioidomycosis[93], and following surgery for a cavity, cavities may recur[101]. Reinfection histoplasmosis has been suggested[16,94], similar to that of tuberculosis.

Cryptococcosis, aspergillosis, candidiasis, and histoplasmosis may co-exist with Hodgkins and other lymphomas, as well as leukemia[10,92,95-100], which complicates the differential diagnosis of hilar adenopathy. Fungi may also co-exist with each other[45].

Other diseases must be differentiated because of similarity in symptoms, x-ray findings, and/or course. These include carcinoma, bacterial and viral pneumonias, pneumoconiosis, sarcoidosis, parasitic infection (especially toxoplasmosis), and the collagen diseases.

Therapy

There is no specific therapeutic agent that is uniformly successful in the therapy of any of the mycoses. Fair results have been reported for the treatment of the pseudofungi *Nocardia* and *Actinomyces* with combinations of sulfonamides, penicillin and the broad spectrum antibiotics. Potassium iodide is used along with other forms of treatment in all of the fungus diseases. However, a warning note should be made referrable to patients who are hypersensitive to the fungus antigen. In these patients, desensitization to the iodide should be made[55] to prevent violent reactions and possible rapid progression of the disease process. 2-hydroxy stilbamidine has been used with some success in actinomycosis, blastomycosis, and oral mucosal histoplasmosis. Resectional surgery is being used more frequently in localized pulmonary lesions in conjunction with rest and chemotherapeutic agents. Progression of disease, however, frequently follows any surgical procedure in sporotrichosis.

North American Blastomycosis, Coccidioidomycosis, and Histoplasmosis

Geographic Distribution

Knowledge of the present and past residence of patients may often be the necessary clue to lead one to the diagnosis of exogenous fungi that have specific areas of endemicity. Although histoplasmosis is considered predominantly limited to the Mississippi, Ohio, and Missouri River Valleys[102-105], new endemic areas have been reported around Lake Champlain and along the St. Lawrence River[32,33], as well as in North Carolina[34] and many other scattered states[35]. Blastomycosis is thought to be concentrated mainly in the Southeastern states[36]. It is also encountered in the Mississippi Valley[37], and isolated cases have been found throughout the United States[38] and Canada[39]. Coccidioidomycoses is rather well limited to the dry, arid areas of the Southwestern United States and Mexico[40-42].

Clinical Findings

Although, as mentioned previously, symptoms and physical findings are not specific, there may be some distinguish-

ing features. Coccidioidomycosis is frequently associated with *erythema nodosum* or *multiforme*[13,43], more commonly in those patients with a high degree of sensitivity to coccidioidin[44]. The primary infection is usually associated with fever and symptoms of an upper respiratory infection or pneumonia.

In histoplasmosis, a disease predominantly of the reticuloendothelial system, hepatosplenomegaly may be a presenting feature, particularly in children. Ninety-five percent of all primary infections are asymptomatic despite a pulmonary infiltration.

Blastomycosis more often has laryngeal and bronchial involvement. Although not a consistent finding, there are usually skin lesions.

X-Ray Findings

Diagnosis cannot be made from an x-ray. In addition to parenchymal involvement, mediastinal adenopathy may be seen in all three[15,16,23]; however, it is more common in blastomycosis and histoplasmosis. Since blastomycosis[15] and histoplasmosis[16] may show enlargement of hilar nodes without apparent evidence of pulmonary infiltration, they must be considered in patients with mediastinal adenopathy. Histoplasmosis is more frequently associated with calcified lesions, and coccidioidomycosis has pulmonary cavitation more often than histoplasmosis or blastomycosis. Cavitation in histoplasmosis is infrequent.

Diagnosis

With the presence of symptoms and x-ray findings of pulmonary disease, fungus disease can be suggested by places of residence, and supported if there are lesions in other organ systems that may be affected by fungi. Presumptive diagnosis can be made by skin tests and/or serologic means. A positive skin test is of the same significance as a positive tuberculin test. It means only a primary infection, at some time in the past, and does not indicate activity. The blastomycin skin test is least sensitive of the three[45,46]. Although the skin test may be negative[47,48] with active disease, it is usually either very early in the infection[13,49] or, as in tuberculosis, an anergy may develop if there is a fulminating disease[13,48,50]. When skin testing is done, all three fungi should be tested at the same time due to a cross reaction that may occur between histoplasmin and coccidioidin[13,51] and between histoplasmin and blastomycin[51-55]. Usually, there is no difficulty in identifying the disease present. The greatest degree of positivity indicates the fungus present. It is not necessary to do serologic tests before skin tests are done since the skin testing material will not evoke humoral antibodies[13,45]. If a positive compliment fixation test for histoplasmosis is already present, the histoplasmin test may cause an increase in titer.

An active disease process is present if there is a positive serologic test, and the change in titer reflects the prognosis of the disease in coccidioidomycosis[56] and blastomycosis[57], but is not as sensitive in histoplasmosis [58]. However, the complement fixation test may be negative[54] in the presence of active disease. In coccidioidomycosis, the precipitin test will appear first[13,54], 50% being positive in the first week and over 90% positive within the first month. This test will usually be negative within two to three months. By this time the complement fixation test is positive, and may persist up to a year after the height of the active infection and occasionally longer, but at a low titer. Complement fixation antibodies may be found in as high as 75% of pleural, ascitic, and spinal fluid, in the presence of pleural, intra-abdominal, or meningeal coccidioidomycosis. The complement fixation test is the only serologic test of value in blastomycosis and histoplasmosis. Here again, due to cross reactions[59], complement fixation tests for all three should be done at the same time.

Definitive diagnosis should be made by the isolation and identification of the fungus. Organisms may be found in the sputa, and since bronchial[11,12] and laryngeal[11,60,61] involvement is relatively common in blastomycosis, bronchial washings or laryngeal smears may be of value. *Histoplasma* dies rapidly in sputa standing in collecting containers. Therefore, sputa should be examined while the material is fresh. In systemic disease, *Blasto-*

myces[11] and *Histoplasma*[58] may be cultured from the bone marrow; and *Coccidioides*[47] and *Histoplasma*[58] may be isolated from blood culture. Although Sabaroud's is the universal media for culturing fungi, some cases may be missed if this media alone is used. At least, in addition, a beef infusion agar[31] should be run.

The pathology of fungus disease is not specific. Caseating tubercles have even been reported in histoplasmosis[28]. In histologic examination of biopsy or surgical specimens for identification of organisms in the tissue, the Hotchkiss-McManus modification of the periodic-acid-schiff stain[58,63] should be employed. Organisms may be missed without stain[64]. It must be reiterated, however, that tissue examination is still, only supplementary to cultural procedures[65].

Therapy

Nonspecific therapy is similar to that of tuberculosis: bed rest and adequate diet together with specific therapy; antimicrobial and surgery where indicated. Surgical resection has been employed successfully in blastomycosis[12,15,66], coccidioidomycosis[13,15,18], and in histoplasmosis[15,67,68], when there is a well localized pulmonary lesion.

Specific therapy leaves much to be desired. Although antibiotics and chemotherapeutic agents[66,69] have been used in blastomycosis, 2-hydroxystilbamidine seems the least toxic, most effective agent available[12,70,71], in conjunction with iodides[45], which should be used in histoplasmosis and coccidioidomycosis as well.

Ethyl vanillate[72,73], despite its only 25-30 per cent margin of safety, has to date been more effective than other agents in the therapy of histoplasmosis. Recently 2-hydroxystilbamidine has been used effectively in a localized oral lesion[108] and may prove successful in pulmonary and generalized disease as well. Although prodigiosin[106,107] is used most frequently in the treatment of coccidioidomycosis, only fair results have been obtained. It has been suggested that perhaps here too, 2-hydroxystilbamidine should be given a therapeutic trial.

Summary

1. Pulmonary fungus diseases are more common than hitherto expected.
2. Endemic areas of exogenous fungi are increasing in geographic scope.
3. A brief discussion of the pulmonary fungus diseases has been presented with special emphasis on the most common exogenous diseases: North American Blastomycosis, Coccidioidomycosis and Histoplasmosis.

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CLINICOPATHOLOGIC CONFERENCE

Presented at Mount Sinai Hospital, Chicago, Illinois

DR. L. FELDMAN, Chairman DR. H. RAPPAPORT, Secretary

Abstracted by DR. L. GOLDMAN

Clinical History

The patient, a 56 year old white male, was admitted to Mount Sinai Hospital on September 23, 1954. The patient had been in good health until one week prior to admission, when while working in a cold room, he noted some "twitching" of the left side of his face and some "trembling of the muscles" on the left side of his mouth. The patient denied any headache, dizziness, or vomiting. An electroencephalogram was taken at that time at Michael Reese Hospital, which showed an irritative focus in the right central parietal region.

Following his discharge from Michael Reese Hospital, the patient again noted "twitching and trembling" of the left side of the face and also some "clumsiness and heaviness" in the left arm. He noticed that he could not perform some fine movements with the left hand (like buttoning up his shirt) and also that things would slip from his left hand, although he did not feel weakness in the hand. On the day before admission, the patient noticed that every time he got out of bed, he would stumble and fall to the left side. He stated that his left leg felt "as if there was too much weight on it." On the date of admission. the "twitching" disappeared, but the "clumsiness" of the left hand and the "heaviness" of the left leg persisted.

There was no history of parasthesias, vomiting, or headache. There were no visual disturbances, impairment of hearing, or vertigo. Past history revealed that surgery had been performed in February, 1954, for a perforated duodenal ulcer. Personal history, systemic review, and family history were non-contributory.

Physical Examination

Physical examination revealed a well-developed, well-nourished white male who appeared to be in no acute distress. The face appeared to be flattened on the left side with a disappearance of the wrinkles on the left side of the forehead. There was a slight deviation of the right bucal commisure. Ears, eyes, nose, and throat were negative. The lungs were clear to auscultation and percussion. Cardiac borders were within normal limits. The second aoritc sound was louder than the second pulmonic sound. Rate was a normal sinus rhythm. There were no murmurs. The liver, spleen, and kidneys were not palpable. There were no abdominal masses or tenderness. The extremities showed a moderate edema. Neurological examination revealed the following positive findings: 1) left hemiparesis, greatest in the face; 2) facial paralysis which disappeared on emotional activity; 3) normal pupils, optic fundi, and ocular movements; 4) weakness of the left upper and lower extremities; 5) absent left abdominal and cremasteric reflexes; 6) positive Babinski on the left.

Erratum

The Editors of *QUARTERLY* regret the publication of a typographical error which appeared in the Clinicopathologic Conference published in the May, 1955 issue on page 136, second column. The statement as published read: "Moreover, since the patient did not have the signs and symptoms of tetany, it was felt that on the basis of the postmortem and antemortem blood clinical studies, plus the clinical findings, acute hypocalcemia could *not* be eliminated as the cause of death." It should have read as follows: "Moreover, the patient did not have signs and symptoms of tetany and we felt that on the basis of the clinical studies of postmortem and antemortem blood, and the clinical findings, we *could* eliminate acute hypocalcemenia as the cause of death."

Hospital Course

A lumbar puncture was performed on September 24, 1954, and revealed a pressure of 160 mm. The fluid was clear and colorless. On September 25, 1954, the patient developed a Jacksonian seizure of the left side of the face which lasted for about five minutes. A pneumoencephalogram was done on September 27, 1954. An operation was performed on September 30, 1954.

Laboratory Data

Examination of the blood on admission showed a red blood cell count of 5.25 million, a hemoglobin of 14.8 grams per 100 cc. or 95%, a color index of .91, a white cell count of 11,000, with 8 per cent stabs, 48 per cent segmented neutrophils, 40 per cent lymphocytes, and 4 per cent monocytes.

Urinalysis revealed a pH of 6.0, a specific gravity of 1.022, negative for protein, acetone, and sugar, with an occasional white blood cell per high power field.

Serum glucose was 88 mg., serum urea nitrogen was 10.0 mg., total protein was 7.35 gm., true albumin 5.20 gm., total globulin 2.15 gm., with a true A-G ratio of 2.48, thymol turbidity of 2.4 units, acid phosphatase of 0.5 Bodansky units, alkaline phosphatase of 3.97 Bodansky units, and a negative cephalin-cholesterol flocculation after twenty-four hours.

Spinal fluid examination on admission showed a pressure of 160 mm. of water, a protein of 96 mg., a true cerebral spinal fluid glucose of 72 mg., and no cells. Serologic examniation of the spinal fluid revealed a negative Wasserman and a 0000000000 gold curve. The Kahn and Kline tests were negative.

Clinical Discussion

*Dr. B. Lichtenstein:** This patient became ill suddenly while working in a cold storage room. The illness was characterized by convulsive movements of the left side of the face, unassociated with headache, dizziness or vomiting. He was taken as an emergency case to the Micheal Reese Hospital where an electroencephalogram revealed an irritative focus in the right central region.

*Attending, Mount Sinai Hospital.

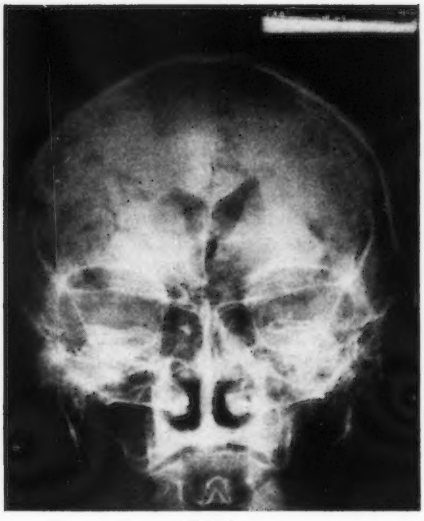


Fig. 1

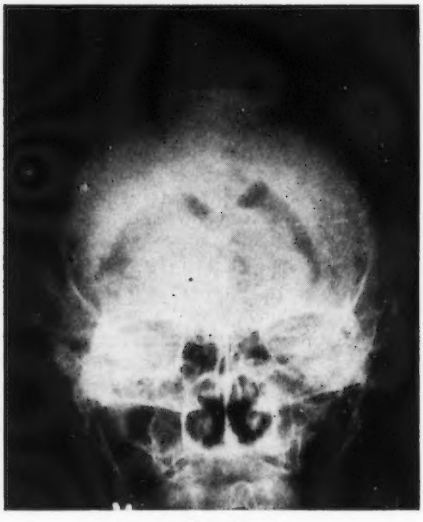


Fig. 2

Figures 1 (left) and 2. Posterior-anterior (left) and anterior-posterior pneumoeucephalograms showing a downward displacement of the right lateral vehicle of about 5 mm. and a diminished distension of the anterior horn of the right ventricle.

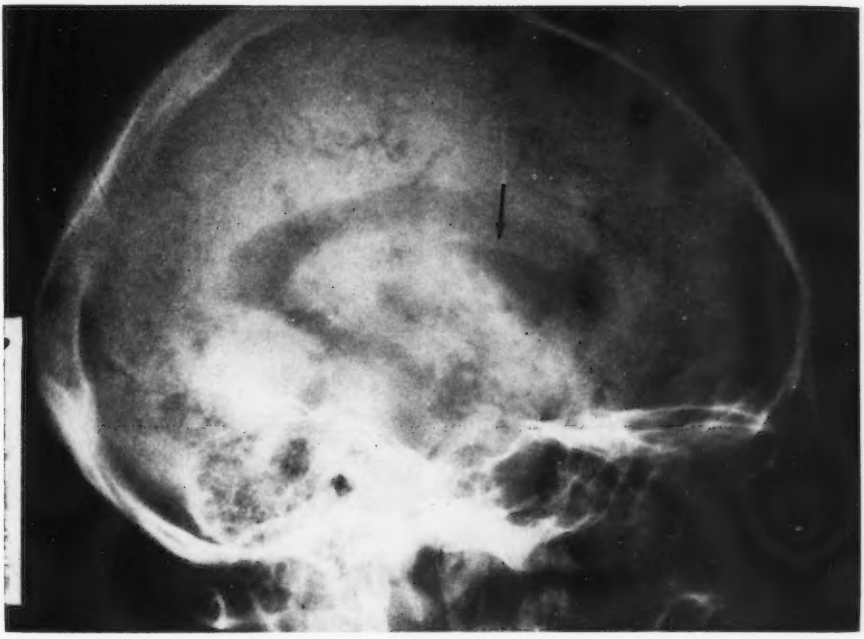


Fig. 3

Fig. 3. Laterial view of pneumoeucephalogram showing evidence of pressure in the right mid-parietal region.

After a few days, he was discharged but the seizures recurred and he consulted Dr. Benjamin Pearlman. At this time, heaviness of the left upper extremity was evident. Since these signs were indicative of organic disease of the brain, he was admitted to the Mount Sinai Hospital for study.

The next day he noticed that he could not perform fine movements with the fingers of his left hand and that he felt as if he was falling to the left. Examination revealed a left hemiplegia but his facial paralysis disappeared when he laughed; in short, it was volitional and not an emotional type of paralysis.

Spinal fluid examination revealed a pressure of 160 mm. of water and a protein of 96 mg. per cent. Routine x-rays of the skull and chest were negative and the fundi did not reveal evidence of increased intracranial pressure. In view of the rapid course, a diagnosis of intracranial tumor was made and an air study by the surgeon revealed a right central lesion. Exploration of this region resulted in the enucleation of a metastatic tumor mass.

Dr. J. Arendt:* (x-ray findings): The flat film taken of the skull on admission showed no bone erosion, but an uncharacteristic, small, well-defined translucency in the left parietal bone. The pneumoencephalogram showed a downward displacement of the right lateral ventricle of about 5 mm. and a diminished distension of the anterior horn of the right ventricle. There is evidence of pressure in the right mid-parietal region suggestive of a tumor pressing from above upon the right ventricle. A lateral shift is not seen and the corpus callosum and the third ventricle remain in the midline.

*Chairman, Dept. of Radiology, Mount Sinai Hospital; Associate Professor of Radiology, The Chicago Medical School.

With the pathological suggestion of the primary origin of the tumor in the kidney, additional films were ordered of the kidneys and they demonstrated the obvious intrarenal tumor which was displacing and pressing upon the upper and middle calyces of the left kidney and behaving like an invasive tumor of the upper, outer pole of the kidney. The bladder, incidentally, is contracted and shows trabeculation suggestive of an additional cystitis.

A chest film taken at that time showed a small, round shadow suggestive of a metastasis. The lateral posterior circumference of the sixth rib on the right side was irregular and suggested an old rib fracture, possibly with an underlying metastasis. Otherwise the lungs were clear and there was no fluid in the pleural cavity.

Dr. B. Pearlman:* The essential problem which arose is one which so commonly occurs; that is, the differential diagnosis between a brain tumor and a cerebro-vascular accident.

*Associate in Medicine, Mount Sinai Hospital.

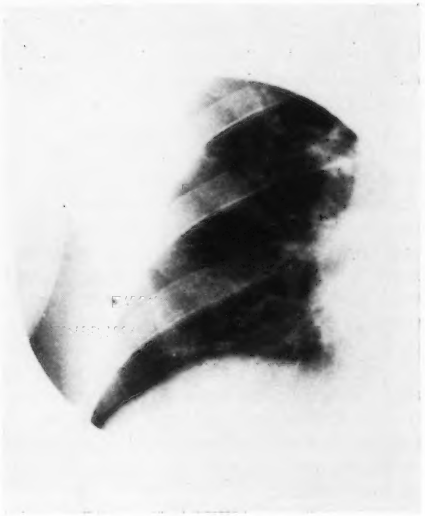


Fig. 4

Figure 4 showing an irregularity of the lateral posterior circumference of the right sixth rib.

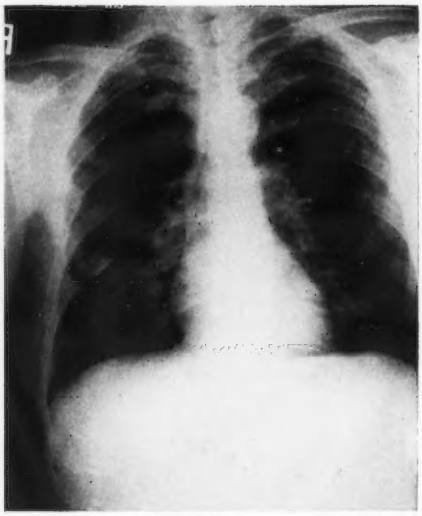


Fig. 5

Chest film suggestive of metastosis. Otherwise lungs are clear.

The primary reason for hospitalizing this patient was to determine the cause of his syncope which he suddenly developed for the first time at work. Upon my first examination, the patient already had some weakness of his left arm and leg. The eye grounds at this time were entirely normal. There was no history of diabetes or hypertension. Upon further observation, we found that these spells of syncope were really episodes of Jacksonian epilepsy involving the left side of his body.

Since the patient did not have any cardiac disease or arrhythmia, any vascular disease such as hypertension or diabetes, and had normal eye grounds as far as vascular disease was concerned, I felt that we were dealing with a cerebral tumor, either a primary or a metastatic lesion. Prostate examination, chest x-ray, careful abdominal examination, serum bilirubin, alkaline and acid phosphatase, blood serology, hematology, and urinalysis were all non-contributory.

I had been treating the patient for a duodenal ulcer for about two years. About four months prior to the discovery of the brain lesion, we found on

routine roentgenographic upper gastrointestinal series to check the status of the patient's duodenal ulcer, a gastric ulcer on the lesser curvature of the stomach. This was very carefully watched with repeated roentgenographic examinations of the stomach every four to six weeks. On careful medical ulcer therapy, the lesion apparently disappeared. Under these circumstances, I felt justified in continuing conservative therapy and to continue to observe the lesion.

At surgery, Dr. M. Tinsley removed a well-circumscribed, localized tumor mass in the right mid-parietal area which the Department of Pathology examined and reported to be of renal origin. Clinically this was not suspected as there were never any symptoms referable to the genito-urinary system. Urinalyses were always negative, and no masses were ever palpated on abdominal examination.

With this pathologic report in mind, I proceeded to order an intravenous pyleogram which definitely showed pathology in the left kidney. Even with this information available, careful reexamination of the patient's abdomen and repeated microscopic examination of the patient's urine failed to disclose any positive findings which might indicate the presence of a renal tumor. Despite the metastatic lesion, I felt that the left kidney should be surgically removed.

Dr. H. Rappaport:* The specimen received by the laboratory consisted of a small spherical mass measuring 1.5 cm. in diameter. This nodule was moderately firm in consistency and on sectioned sur-

*Pathologist, Mount Sinai Hospital; Associate Professor of Pathology, The Chicago Medical School.

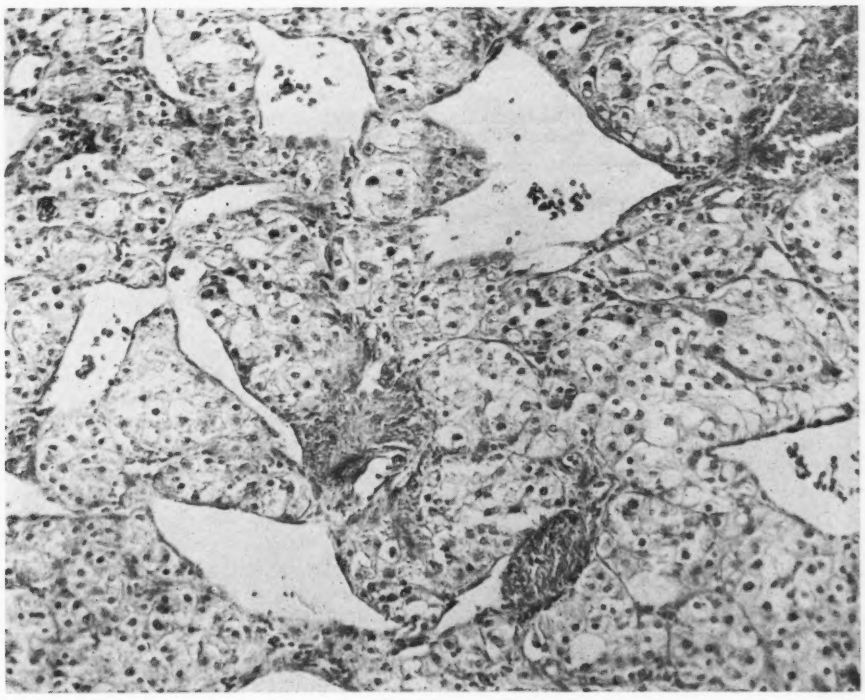


Fig. 6

Figure 6. Microscopic section (260X) of metastatic mass showing the characteristic picture of a renal carcinoma of the hypernephroid type.

face, had a reddish-brown color with foci of necrosis. The lesion was well circumscribed, but not encapsulated. It was entirely surrounded by a thin rim of white brain tissue. Microscopic sections showed the characteristic picture of a renal carcinoma of the hypernephroid type (fig. 6).

The tumor was composed of large polyhedral cells with pale pink or clear cytoplasm and distinct cell borders. Within the tumor, there were many thin-walled sinusoidal blood vessels composed of only a single layer of endothelial cells.

This picture was interpreted as being highly characteristic of a tumor of renal origin and we reported it as a metastatic carcinoma, hypernephroid type, consistent with a primary carcinoma of the kidney.

Dr. L. Maslow:* As you just heard, the diagnosis of renal tumor was made, in effect, by the Department of Pathology, when they reported their histologic findings of the surgical specimen. Thereupon, an intravenous urogram revealed pathology of the left kidney.

Attending, Mount Sinai Hospital; Associate Professor of Urology, The Chicago Medical School.

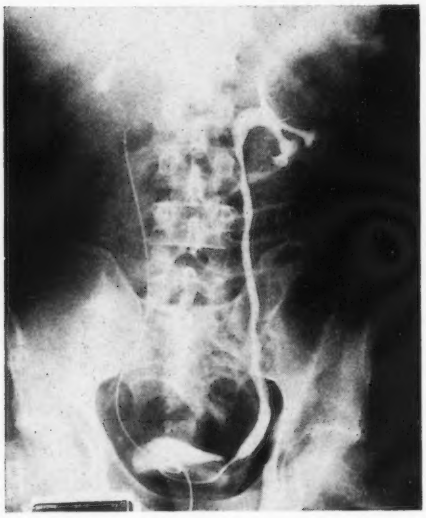


Fig. 7

Figure 7. Note the intrarenal mass displacing the upper and middle calyces of the left kidney.

A complete urologic study was ordered at this time. As on repeated earlier examinations by Dr. Pearlman, the kidneys were not palpable on abdominal examination. Repeated urinalyses were once again entirely negative. The patient did not feel any pain or tenderness in the flank. A retrograde pyelogram confirmed the diagnosis of tumor of the left kidney.

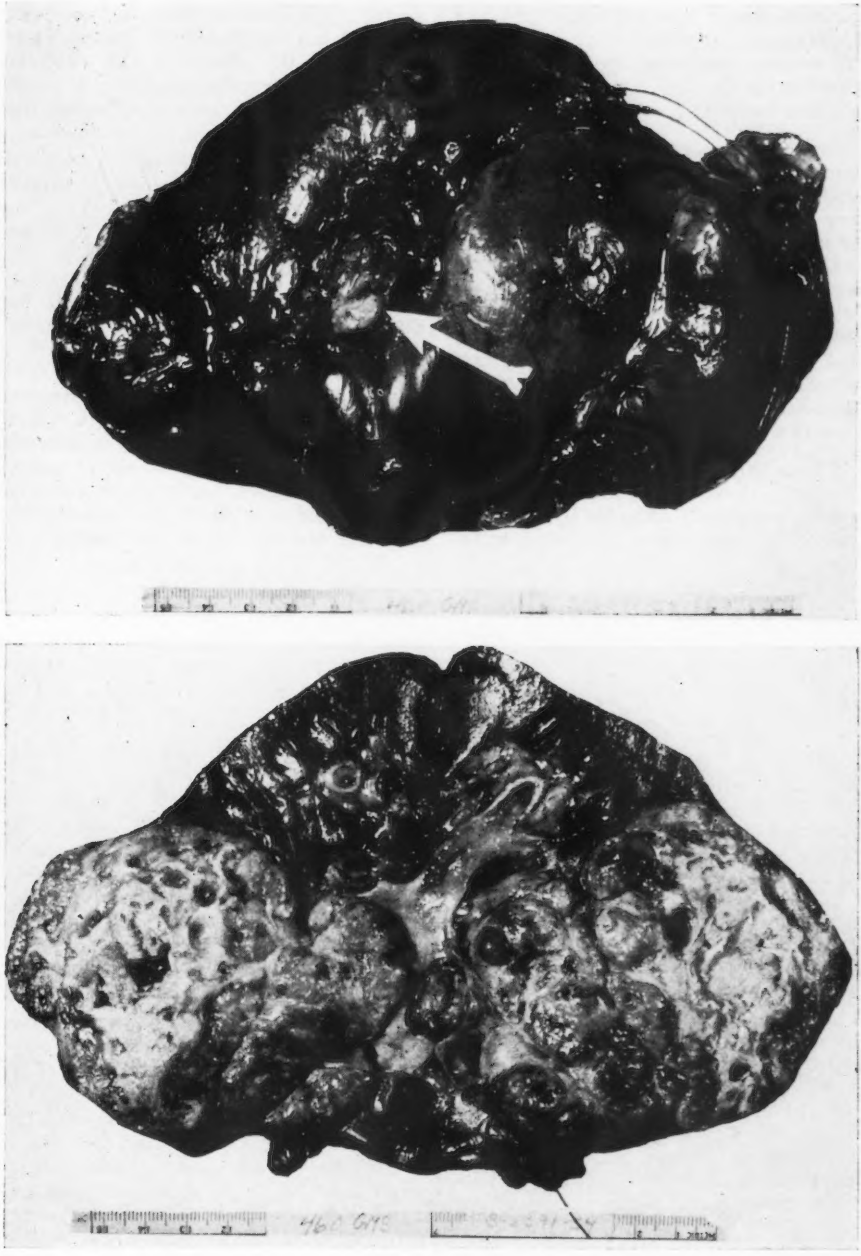
A left nephrectomy was performed through a lumbar approach. A large tumor was found involving the upper pole of the left kidney. The entire mass was found under the lower ribs. This explains why the kidney was not palpable clinically. A finding of bad prognostic omen was the demonstration of the invasion of the renal vein by tumor tissue. The patient recovered from the operation without incident, only to die two months later of cachexia and general debilitation.

Kidney tumors will, in rare instances, remain entirely silent until they metastasize, either to the brain, lung, or bone. This patient presented himself with the symptoms of cerebral metastasis. In this case there were metastases to both the brain and lungs.

The main point of interest to me, after I was called in for consultation, was whether the primary tumor of the kidney should be removed surgically or should the patient be left alone in view of the poor prognosis of hypernephroid carcinoma. There have been a few reports of cases in the literature where, after removal of the primary tumor of the kidney, the lung mastatases disappeared with x-ray therapy.

One report cites a case where nephrectomy was performed for the primary tumor and a subsequent pneuonectomy was performed for a solitary lung metastasis with a successful result. However, these instances are extremely rare. Nevertheless, families of the afflicted patients hear of such remote possibilities of hope and they insist on surgery.

In another similar case, a woman first presented herself with the chief complaint of pain in the ribs. A biopsy of the rib disclosed on histologic section a hypernephroid carcinoma. Urologic study then revealed a tumor of the kidney which, upon the insistence of the



Figs. 8 and 9

Figures 8 and 9 (Bottom). Gross specimens of left kidney removed at surgery.

patient's family, was removed surgically. Here again, the patient made an uneventful recovery from the operation, but succumbed six months later from metastases and cachexia.

Dr. H. Rappaport: The specimen submitted by Dr. Maslow was a left kidney with a segment of the ureter. The kidney weighed 460 grams and measured 12 x 10 x 7.5 cm. There was a large tumor mass measuring approximately 9 x 8.5 x 7 cm. It occupied the upper half of the kidney. On sectioned surface, this mass appeared to be roughly spherical and had a variegated appearance with alternating areas of yellow tumor tissue, foci of necrosis, and cyst formation (fig. 9).

The renal pelvis did not appear to be invaded. However, the renal vein was almost completely occluded by tumor tissue. The term tumor thrombus is frequently employed to designate the presence of an intravascular tumor mass molded to the shape of the vascular lumen. As illustrated in figure 8, surgical resection had to be made through the tumor tissue occluding the renal vein.

While the pelvis was compressed and deformed, it was not invaded by tumor. Lack of invasion of the kidney pelvis explains the absence of urinary findings. If the renal vein is invaded before the renal pelvis, then we may have a situation in which the metastases occur before abnormal urinary findings are present.

It has been pointed out by Dr. Maslow that renal carcinoma of the hypernephroid type is one of the tumors in which some observers believe that the presence of a solitary metastasis is not a contraindication against nephrectomy and in which removal of both the primary tumor and the metastasis is advocated.

SCHOOL NOTES AND NEWS

FACULTY APPOINTMENTS

Dr. John J. Sheinin has announced the following appointments to the faculty of The Chicago Medical School:

Department of Medicine

Dr. Carl Weiner as Clinical Assistant.

Department of Obstetrics and Gynecology

Dr. Frank E. Rubovits as Clinical Assistant Professor.

Department of Neurology and Psychiatry

Mr. John B. McAllister as Clinical Assistant in Psychiatry.

Department of Pediatrics

Dr. Irene Kuras as Clinical Assistant.

Department of Surgery

Dr. Jerome J. Moses as Clinical Associate.

Dr. Sheinin has announced the following faculty promotions:

Department of Dermatology and Syphilology

Dr. David M. Cohen, Acting Chairman of the department, has been appointed to full chairmanship.

Department of Medicine

Dr. George C. Coe to Clinical Associate Professor.

Dr. Irwin Dvore to Clinical Instructor.

Dr. John Horkavy to Clinical Instructor.

Dr. Harold R. Kamenear to Clinical Instructor.

Dr. Solomon L. Pearlman to Clinical Instructor.

Dr. David B. Radner to Clinical Associate Professor.

Dr. Irving H. Zitman to Clinical Assistant Professor.

Department of Orthopedic Surgery

Dr. Allan B. Hirschtick to Clinical Assistant Professor.

Dr. Irving Wolin to Clinical Associate Professor.

Department of Neurology and Psychiatry

Dr. LeRoy P. Leavitt to Assistant Professor.

ALUMNI NEWS

Class of 1935

Congratulations to Dr. and Mrs. Robert Adelman on the birth of a son, Corey Brian, on November 8, 1954.

Class of 1943

Dr. Bart D. Iaia has announced the opening of his office at 164 State Street, Auburn, New York, for the practice of general surgery and general thoracic surgery.

Class of 1944

Dr. Bernard Tumarkin has announced the opening of his office at 220 Miracle Mile, Coral Gables, Florida, for the practice of neurology and psychiatry.

Class of 1948

Dr. Theodore Cohen has announced the opening of an office for the practice of internal medicine at 110-45 71st Road, Forest Hills, New York.

Dr. Sophia A. Joffe has announced the opening of an office on Roosevelt Road, Alternate U. S. 30, West Chicago, Illinois, for the practice of general medicine.

Class of 1949

Dr. Anthony C. Milea has announced the opening of his office at 119 West 11th Street, New York City, for the practice of obstetrics and gynecology.

Class of 1950

Dr. Herbert L. Fishbein has announced his association with the department of radiology of the Lakeside Medical Center, 987 East Jefferson Avenue, Detroit, Michigan.

Dr. Louis L. Lunskey has published a recent article, "Murderous 'Acting Out' as a Primitive Defense to Master Anxiety", in the *American Journal of Psychotherapy*, IX:2, April 1955. An abstract of this article appeared in *QUARTERLY*, Volume 16, Number 3.

Class of 1951

Best wishes to Dr. Suzanne Allen Widrow and Dr. Lee Widrow on the birth of their daughter, Carol Anne, on January 2, 1955.

Class of 1952

Dr. Arnold Grier has announced the opening of his office for the practice of internal medicine at 350 Lincoln Road, Miami Beach, Florida.

Class of 1953

Congratulations to Dr. and Mrs. Jerome Handler on the birth of their son, Robert Paul, on April 30, 1955. Dr. Handler is a resident in internal medicine at the West Side Veterans' Administration Hospital in Chicago.

Class of 1955

Congratulations to Dr. Stanley Becker on his marriage to the former Miss Sandra Orth, of Evanston, Illinois, on June 19, 1955.

Best wishes to Dr. and Mrs. Jack Gilman on the birth of their daughter, Rose Diane, on April 28, 1955.

Best wishes to Dr. and Mrs. Eric Saltzman on the birth of their son, Robert Bruce, on February 26, 1955.

Congratulations to Dr. Sheldon Waldman on his engagement to Miss Ruth Forkosh of Chicago, Illinois.

Best wishes to Dr. Howard Weinstein on his marriage to the former Miss Sara Goldsmit, of Mexico City, Mexico, on June 26, 1955.

STUDENT NEWS

Class of 1956

Best wishes to Irwin Reichman of Averne, N. Y., on his engagement to Miss Sandra Miller of New York, N. Y., on June 27, 1954.

Class of 1958

Best wishes to Joel Curtis on the occasion of his marriage to the former Miss Adrienne Wincor of Brooklyn, New York, on March 26, 1955.

Best wishes to Jay Epstein on the occasion of his marriage to the former Miss Harriett Levinson of Pittsburgh, Pennsylvania, on June 19, 1955.

Best wishes to Clifford Rubin on the occasion of his marriage to the former Miss Edythe Reitman of Chicago, on June 25, 1955.

Congratulations to David Heinrich on his engagement to Miss Gladys Muskal of Chicago, Illinois, on June 27, 1955.

Congratulations to David Woldenberg on his engagement to Miss Rita Grabino of Brooklyn, New York, on July 17, 1955.

Congratulations to Richard Rubin on his engagement to Miss Lucille Diamond of Brooklyn, New York.

ORGANIZATION NEWS

Phi Delta Epsilon

Beta Tau Chapter of Phi Delta Epsilon extends their heartiest congratulations to the 46 new members initiated at the annual Tri-Chapter Dance on February 19, 1955. The members of our chapter wish to thank our Chicago Alumni Club who sponsored this memorable dinner-dance which was held at the Furniture Club.

The newly elected officers for 1955-56 are: President, Burt Garfinkel; Vice President, Sandy Kornblum; Secretary, Eli Schessel; Treasurer, Arnold Serbin; Assistant Secretary, Joseph Ansfield; Assistant Treasurer, Irv Tracer; Convention Delegate, Ronald Lieberfarb; and Student Council Representative, Ted Balsam.

The Memorial Day week-end was highlighted by the Senior Dinner Dance held at the Tam O'Shanter Country Club. More than 160 students and alumni enjoyed the evening which was dedicated to our 32 graduating seniors, each of whom received a pin which will serve both as a reminder of the Chicago Medical School and of the fraternity.

We are looking forward to this year's social and educational program. Dr. Harry Rolnick, long time member of Phi Delta Epsilon, has been chosen to be honored at this year's lectureship series on October 31, 1955.

Phi Lambda Kappa

During the past few months the activities of Phi Lambda Kappa Fraternity have been limited to the planning of the Annual Senior Dinner-Dance which was held on May 28, 1955 at the Sirloin and Saddle Club of the Stockyard Inn. At this very successful dinner-dance, which was attended by over one hundred fraters and alumni, the nineteen seniors of Alpha Rho were presented with their Senior Keys by our faculty advisor, Dr.

Joseph Poticha. The members of the fraternity, in turn, presented Dr. Poticha with a Local Alumni Award Key as a token of our gratitude for his devotion to our chapter during the past years.

The presentation of the Annual Alpha Rho Gold Medal Award was also made at this affair. This year the Gold Medal was presented to the Reverend John C. Evans, Religion and Education Editor of the Chicago Tribune and Vice-Chairman of the Board of Trustees of The Chicago Medical School, for his loyalty and devotion to our school.

On April 26, 1955, the Alpha Rho Chapter held its election of officers for the coming year. The following fraters were elected:

President, Peter Brodney; Vice-President, Sanford Cole; Treasurer, Herbert Aronson; Recording Secretary, Leslie Malkin; Alumni Secretary, Martin Salzman; Historian, Stuart Freifeld; Student Council Representatives, Peter Berczeller and Daniel Ludwig; Corresponding Secretary, Marcel Horowitz.

QUARTERLY

The Senior Editorial Board of *QUARTERLY* wishes to announce the election of Martin Salzman ('56) as Editor-in-Chief of *QUARTERLY;* Jesse Berkowitz ('56) as Managing Editor; Sherwyn E. Warren ('56) as Medical Editor; and Jesse S. Gochman ('56) as Features Editor.

"LOST ALUMNI"

Do you know where the following alumni of The Chicago Medical School can be located? *QUARTERLY* and the Alumni Office has lost track of these graduates. If you have any information as to the present address of these alumni, the Editor of *QUARTERLY* will be grateful for your assistance in locating them. Address your letters to: The Editor, The Chicago Medical School *QUARTERLY*, 710 S. Wolcott Avenue, Chicago 12, Illinois.

Class of 1916
- Algoth, Ernst A.
- Jahp, Minnie
- McKenzie, Prentiss
- Morrison, W. J.
- Ouda, Ignatius
- Ramirez, Jose M.
- Storgaard, H.
- Strunk, Alois C.
- Wotonabe, I.

Class of 1917
- Fellars, Edgar
- Foty, Saviour R.
- Gorov, Ida R.
- McDade, Robert
- Minas, Yervant
- Seidner, Maurice L.
- Sonnenfeld, Frederick E.
- Strange, Tieghman B.
- Stuttaford, Mina Ross
- Trice, Jesse Sylvander
- Wood, Dorr E.
- Yaffe, George

Class of 1918
- Arens, Robert A.
- Hoefle, Edward
- Igoe, Edward J.
- Kilberg, N. A.
- McFarland, Lee
- Mahnteaux, E. C. D.
- Marin, Ben
- Martin, Joseph I.
- Morrison, John
- Percival, James S.
- Schmoll, Fritz W.
- Schneider, Albert L.
- Talbot, Melvin C.
- Teed, Edward Harmon
- Wanner, Jay George
- White, W. Wallace

Class of 1919
- Mendoza, Simon F.
- Montero, E. A.
- Rachelis, M. R.
- Salesi, L. O.

Class of 1920
- Ferendez, Alfred Z.
- Meixueiro, Samuel G.
- Miller, Otto H.
- Minderout, Will J.
- Monkiewicz, Sigmund
- Ranala, Marcellano R.
- Roberts, Oscar F.
- Robinson, Edward J.

Class of 1921
- Barrier, Gustave A.
- Covington, Cyrus B.
- Lazatin, Mariano
- Lee, George N.
- Santos, Carlos
- Wilson, Mrs. Mary

Class of 1922
- Baier, Augustus
- Gelder, Mark S.
- Gibson, Carl Chesney
- Hoffman, Isadore I.
- Jorgenson, Thurstong
- Radway, Samuel
- Rathbun, Maten F.
- Riego, Raymond C.
- Rivero, Armado Jasto
- Troyer, George D.

Class of 1923
- Campbell, William R.
- Cortesi, Dante
- Davis, Raymond Hill
- Naryanckas, Victor
- Ross, L. J.
- Turbow, A. O.
- Waring, Mary Fitzbutler

Class of 1924
- Anderson, Ernest L.

Class of 1925
- Anderson, Jesse J.
- Burda, Stanley Walter
- Crocus, Adam
- Dispensa, Rose Mary
- Krakowski, Julian P.
- Labash, Charles
- MacCormack, Juan Jose
- Marcus, Marion Esther
- Megahy, James A.
- Melendez, Juan
- Mueller, George E.
- Negishi, Ryohei
- Novack, Louis
- Nygood, Abraham
- Secunda, Herman H.

Class of 1926
- Gram, Gunnar
- Pieczynski, A. Stanley

Class of 1927
- Akers, E. David
- Carrera, Oscar G.
- Clark, Homer Maxwell
- Cruzat, Roscoe M.
- Drummond, Dolores
- Kesling, Floyd J.
- Klein, Harry A.
- Marquez, Felix
- Townsend, C. Lambert
- Whamond, Robert F.

Class of 1928
- Duerme, Francis M.
- McKinney, Timothy
- Susser, Max H.
- Tidleary, Louis
- Zin, Stanley A.

Class of 1929
- Gore, George J., Jr.
- Hayverde, Beresford
- Shapiro, Joseph

Class of 1930
- Schroeder, William
- Tse, Wing Yan

Class of 1931
- DeLeon, Benito C.
- Lawn, Hugh E.

Class of 1932
- Jacobs, Herbert
- Kenny, William J.
- Solbrig, Charles Rudolph
- Suino, John Baptist

Class of 1933
- Altbach, George Louis
- Brodsky, Frank
- Cohen, Leon H.
- Ellis, J. Gilbert
- Hosman, I. Daniel
- McClure, George Malcolm
- Mullen, Vincent Victor
- Tyllas, Harry A.
- Zablocki, George Henry

Class of 1934
- Bartolucci, Raymond J.
- Grezda, Jane L.
- Herbert, William J.
- Krajec, Andrew
- Kucharski, Stanley C.
- Lynch, James I.
- Mirikitani, Isami
- Porter, John T.
- Stephens, Jerry H.

Class of 1935
- Batko, Bernard B.
- Bissell, John H.
- Gallagher, George C.
- Hall, Philip V.
- Hennessey, James J.

Hirchfield, Stanley Adrian
Kapusinski, Frances B.
Mayfield, Ike J.
Newton, William A.
Walker, John V.

Class of 1936
Gilula, Adolph
Hecht, Morris Ralph
McKay, Hayden Edwards, Jr.

Class of 1937
Balles, Edward S.
Cervera, Dominic Louis
Orlando, William Fazio
Rettinger, Leo Martin

Class of 1938
Berger, Earl Robert
Digal, Leonilo Tunampus
Grizzaffi, Anthony Louis
Hays, Jack D.
Lewis, Louis L.
Rosenbloom, Joseph
Yosko, Oscar Norman

Class of 1939
Berman, Peter
Cooper, Samuel S.
Hart, Leon Martin
Pokorski, Annette J.
Scanlan, Nestor
Shoger, Gilbert
Smith, Paul Edward
White, Edward

Class of 1940
Barbour, Ben Harold, Jr.
Comarr, Avrom Estin

Class of 1941
De Quevedo, Eugenio Garcia
Grzybowski, Stanley Theodore
Howland, Bernard Francis
Leach, Charles Lewis, Jr.
Metry, John Michael
Swartz, Herbert Porter
Ventimiglia, Anthony J.

Class of 1942
Geller, Garrison
Schrag, Harry J.
Terrell, Woodrow La Mance

Class of 1943
Bendet, Samuel S.
Berman, Joseph Samuel
Douglas, John Evans
Futernick, Benjamin
Gelbin, Ruehl Milton
Lazarus, Sydney Simon
Light, Arthur
Meyers, Lawrence H.
Moyers, Roald Allen
Penner, Bernard Jerome
Permesly, Harry Michael
Raicus, Eugene
Sanders, Roland C.
Schreiber, William
Slepikas, Victor Paul
Uyeno, Frank Isamu
Wallace, George
Wein, Eber

Class of 1944
Bodwin, Robert Edward
Camp, Turner
Curtis, Clayton Ross
Ehle, Jack Friedland
Felderman, Ephraim Jacob
Mosak, Harold
Rothman, Stanley David

Class of 1945
Cusack, Patrick James
Dale, David Oscar
Dansby, Garland Lee
Elmer, Russell Edwin
Friedman, Stanley
Kaiser, Murray L.
Kretchmar, Howard Lyle
Lerner, Marvin
Morrison, Donald Dov
Rockliff, Burton Wolfe
Rodney, Marvin Benjamin
Sloan, Donald Evans
Small, Harry A.

Class of 1946
Bookspan, Saul C.
Gerber, Jerome
Housberg, Mortimer
Jaffe, Edward Bertram
Liewen, Benedict Egon
Mintzer, Sidney
Polskin, Louis J.
Rogers, Eugene Jack
Siegel, Alan Cyril
Silverstein, Murray Herman
Ziporyn, Marvin Charles

Class of 1947
Batlan, Lawrence E.
Bloom, Bernard
Bucar, John Robert
Goldy, Melvin Leonard
Helfer, Sidney Paul
Levine, Seymour
Raubitschek, Howard Allen
Rockowitz, Erwin H.
Simner, Robert Roy
Thompson, Carl Winston
Weinstein, Arnold Roy
Zingher, Henry C.

Class of 1948
Brown, Caroline Winograd
Brown, Harold Nathan
Chang, Kenneth Yun En
Feinstein, Sherman C.
Houda, Allan Joseph
Kassel, Arthur David
Kayman, George
Leichtling, Melvyn
March, Harold William
Migdal, Elliot
Platt, Daniel W.
Salzman, Wallace Laurence
Schlotterbeck, Miriam
Schrenzel, Alan
Skaggs, Frank Pryor, Jr.

Class of 1949
Justen, Jerome William
Kaplan, Seymour Herman
Rosenstein, Abraham Sydney
Schaffner, Morton Jack
Singerman, Leonard

Class of 1950
Guido, John Augustuvus
Lieberman, Murray
Packer, Marvin S.
Ravich, Lawrence
Rosner, Sol
Schuler, W. S.
Silberman, Jack

Class of 1951
Eisenberg, Melvin
Friedman, Franklin Paul
Kahn, Joseph Louis
Meyers, Chester Lionel
Perl, Frederick Louis
Pick, Melvin M.
Schiff, Samuel Barrett
Turkewitz, Hyman
Udkoff, Claude

Class of 1952
Behrman, Jack
Bernstein, Aaron M.
Blass, Norman Herbert
Gragman, Robert Donald
Finkel, Marion Judith
Krasner, Bernard
Loeb, Sheldon
Rangell, Nelson
Rosenberg, Murray Kenneth

Class of 1953
Arons, Ernest
Rolnick, Donald Trace

THE CHICAGO MEDICAL SCHOOL
INTERNESHIPS

Class of June, 1955

Eugene Allen
Kings County Hospital
Brooklyn, New York

Edward Altchek
Long Island Jewish Hospital
Long Island, New York

Stanley C. Becker
Jewish Hospital
St. Louis, Missouri

Herbert Bengelsdorf
State University of Iowa Hospitals
Iowa City, Iowa

Burton T. Blackman
Cook County Hospital
Chicago, Illinois

Ronald Blatt
Los Angeles County Hospital
Los Angeles, California

Herbert A. Blough
Cincinnati General Hospital
Cincinnati, Ohio

Seymour Bradus
Cook County Hospital
Chicago, Illinois

Arthur Brandt
Beth El Hospital
Brooklyn, New York

Philip Brodsky
Maimonides Hospital
Brooklyn, New York

Arnold W. Brody
Cook County Hospital
Chicago, Illinois

Norman G. Brunner
Kings Couny Hospital
Brooklyn, New York

Jeanette H. J. Chang
Cook County Hospital
Chicago, Illinois

Benjamin Cohen
Kings County Hospital
Brooklyn, New York

Marvin B. Cohen
Veterans Administration Hospital
Los Angeles, California

Bernard Deitch
Memorial Hospital
Wilmington, Delaware

Stuart M. Eichenfield
Syracuse Medical Center
Syracuse, New York

Stanley H. Enker
Jewish Hospital
Brooklyn, New York

Maurice B. Fields
Mount Sinai Hospital
Cleveland, Ohio

Jack Gilman
Milwaukee County Hospital
Milwaukee, Wisconsin

Howard J. Goldman
Beth Israel Hospital
New York, New York

Mitchell B. Goldman
Kings County Hospital
Brooklyn, New York

Jerome Goldstein
Albert Einstein Medical Center
Southern Division
Philadelphia, Pennsylvania

Walter C. Goldstein
Philadelphia General Hospital
Philadelphia, Pennsylvania

Melvin G. Goldzband
Cook County Hospital
Chicago, Illinois

Milton Greenberg
Hackensack Hospital
Hackensack, New Jersey

Melvin Greenblatt
Mount Sinai Hospital
Cleveland, Ohio

Jason I. Greenstein
Rhode Island Hospital
Providence, Rhode Island

Sheldon Gross
Kings County Hospital
Brooklyn, New York

Philip J. Gutentag
Mount Sinai Hospital
Cleveland, Ohio

Marvin I. Herz
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Chicago, Illinois

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Kings County Hospital
Brooklyn, New York

Irwin H. Krasna
The Mount Sinai Hospital
New York, New York

Mortimer J. Lacher
Beth Israel Hospital
New York, New York

Gerard A. Levi
Jewish Hospital
Brooklyn, New York

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Los Angeles County Hospital
Los Angeles, California

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Beth Israel Hospital
New York, New York

Robert Margid
Cook County Hospital
Chicago, Illinois

Phillip I. Mozer
Cook County Hospital
Chicago, Illinois

Jordan M. Rhodes
Cook County Hospital
Chicago, Illinois

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San Francisco Hospital
San Francisco, California

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Kings County Hospital
Brooklyn, New York

Sheldon P. Rothenberg
Maimonides Hospital
Brooklyn, New York

Eric I. Saltzman
Maimonides Hospital
Brooklyn, New York

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Kings County Hospital
Brooklyn, New York

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State University of Iowa Hospitals
Iowa City, Iowa

Alicia Schwieger
Michael Reese Hospital
Chicago, Illinois

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University of Illinois Research and Educational Hospitals
Chicago, Illinois

Melvin Silverman
Harbor General Hospital
Torrance, California

Herbert Sohn
Bellevue Hospital Center
Third Medical Division
New York, New York

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Philadelphia General Hospital
Philadelphia, Pennsylvania

Aaron L. Southren
The Mount Sinai Hospital
New York, New York

Helmuth A. Stahlecker, Jr
Cook County Hospital
Chicago, Illinois

Lawrence Strenger
Hospital of St. Raphael
New Haven, Conencticut

Seymour Stricker
Flushing Hospital and Dispensary
Flushing, New York

David Tuman
Kings County Hospital
Brooklyn, New York

George H. Veldstra
Los Angeles County Hospital
Los Angeles, California

Sheldon Waldman
Long Island Jewish Hospital
Long Island, New York

Howard Weinstein
Long Island Jewish Hospital
Long Island, New York

Charles L. Weisenthal
Milwaukee County Hospital
Milwaukee, Wisconsin

Ernest M. Weitz
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Long Island, New York

Morton Wexler
Los Angeles County Hospital
Los Angeles, California

Burton Zeiger
Philadelphia General Hospital
Philadelphia, Pennsylvania

Lawrence H. Zingesser
Graduate Hospital of the University of Pennsylvania
Philadelphia, Pennsylvania

BOOK REVIEWS

A TEXTBOOK OF MEDICINE. Edited by Russell L. Cecil, M.D. and Robert F. Loeb, M.D. Ninth edition. Cloth. 1786 pages. Philadelphia: W. B. Saunders Co., 1955. $15.00.

The editors of this new edition state that they are trying to reflect the many advances in the field of internal medicine that have taken place in the four years since the eighth edition went to press. Emphasis is on the correlation of the physiologic and biochemical aspects with the clinical description of the disease entity.

The ninth edition contains the following articles on subjects which have not been covered in previous editions: Colorado Tick Fever, Coxsackie Viral Infections, Epidemic Hemorrhagic Fever, Cat Scratch Disease, Acute Infectious Nonbacterial Gastroenteritis, an introduction to Bacterial Diseases, *Hemophilus ducreyi* Infections, Nonsyphilitic Treponematoses (Bejel, The Leptospiroses Pretibial Fever, Leptospiral Meningitis, Grippe-like Illness and other forms of Leptospiral Infections), Visceral Larva Migrans, Dirofilariasis, Milk Sickness, Scleroderma, Blast Injury, War Gases, Burning Feet Syndrome, Pyridoxine (Vitamin B_6) Deficiency, Vitamins and Blood Regeneration, Kwashiorkor, Atherosclerosis, Xanthomatosis, Dehydration and Fluid Balance, Melanosis, Carotenemia, Portal Hypertension, Pulmonary Arteriovenous Fistula, Cystic Disease of the Lungs, Asbestosis, Acute Nonspecific Pericarditis, Senile Heart Disease, Introduction to Diseases of the Muscles, The Painful Shoulder (Calcific Tendinitis, Adhesive Peritendinitis, Arthritis of the Shoulder, Shoulder-Hand Syndrome), Hyperostosis Frontalis Interna, Hemiplegia, Delirium, The Dementias, Nonpurulent Meningitis, Developmental Anomalies of the Cervicomedulary Juncture, and Petrositis. This text will be of great value to the clinician and student alike.

* * * *

CLINICAL BIOCHEMISTRY by Abraham Cantarow, M.D. and Max Trumper, Ph.D. Cloth. Fifth Edition. Philadelphia and London: W. B. Saunders Co., 1955. $9.00.

The authors have completely revised this text and have added expanded discussions in such vital fields as liver function, kidney function, plasma protein abnormalities, biologic significance of nucleic acids, uric acid metabolism porphyrin metabolism, iodine metabolism, lipoproteins, fatty liver, potassium metabolism, acid base balance, and the evaluation of endocrine function, especially that of the thyroid and adrenal. The text eliminates discussions of any aspects of biochemistry not applicable to the clinical diagnosis of diseases. No charts listing abnormalities of specific pathologic entities are given as in previous editions. References are as late as 1954. The text is an excellent source of instruction in clinical biochemistry and pathophysiology and is meant to be instructive for the clinician already grounded in basic sciences.

Specific biochemical abnormalities are listed in detail in the index and thereby serve as a differential diagnosis for quick reference. In this manner, the authors expect to aid the reader in rapidly locating those sections of the text wihch might help him in evaluating the significance of the specific pathological findings. The advanced student of medicine might well find the text a valuable condensation of those salient aspects of human biochemistry which are of importance in disease.

* * * *

THE PRACTICE OF DYNAMIC PSYCHIATRY by Jules H. Masserman, M.D. Cloth. First Edition. 790 pages. Philadelphia: W. B. Saunders Co., 1955. $12.00.

This book extends the applications of biodynamics set down nearly a decade ago by the author in *Principles of Dynamic Psychiatry*, to clinical psychiatry and to the theory and practice of medicine and its specialties. The information it contains is balanced as to the theoretical and practical aspects of the subject. The first four sections form a firm foundation for the fifth which is devoted to clinical therapy.

In addition to covering the standard material found in most texts, one finds many of those important but unwritten topics in this book. Chapters on such subjects as narcosis, hypnosis, alcoholism, and forensic psychiatry help make the book an extremely practical work for students, interns, residents, and practitioners alike.

Of special interest to students are the sections on history and development of different schools of analysis, technique of interviewing and principles of psychiatric examination.

* * * *

GENERAL ENDOCRINOLOGY by C. Donnell Turner, Ph.D. Cloth. Second Edition. 553 pages. Philadelphia: W. B. Saunders Co., 1955. $8.00.

The viewpoint of this text is that of endocrinology as a basic biologic scence rather than as a clinical science. Therefore, it should serve the student and clinician as a good reference to the basic science of endocrinology. The author does not deal extensively with the clinical aspects of the subject.

All chapters and bibliographies have been thoroughly revised and brought up to date since publication of the first edition in 1948. In view of the tremendous amount of research published since 1948 on the adrenal cortex and pituitary gland, these sections have been practically rewritten. A new chapter has been added on "Adaptive Reactions to Stress."

The material dealing with "Neurosecretion" has been amplified and expanded, particularly with reference to the concept that neurosecretory cells connect the nervous and endocrine systems. This theory advances a tentative explanation for the manner in which nervous excitations may be translated into hormonal activity; an explanation which may have broad implications in the fields of psychology and psychosomatic medicine.

All chapters are divided into sections on the anatomy (gross, microscopic, developmental and comparative), the biochemistry, and the physiology of the gland in question.

* * * *

PATHOLOGY FOR THE SURGEON by William Boyd, M.D., F.R.C.S., F.R.C.P., M.R.C.P., F.R.S., D.Sc. Cloth. 737 pages. and 547 illustrations. Philadelphia: W. B. Saunders and Co., 1955. $13.50.

To the first complete revision of a well known text which has gone through five previous revisions and 19 English printings, there has been added a new feature, that of a correlation between the pathologic and clinical features. It is a very welcome addition. In keeping with the shift of emphasis from the traditional presentation, the title has also been changed.

The work, as in previous editions, is full of succinct and pithy quotations from many ages and cultures together with the author's own unique comments, some of which are used in attempts to force acceptance of his views on controversial matters, some to emphasize an important point, and others to increase the reader's interest in the subject. Other unfortunate remarks, such as this quotation from page 415, concerning the treatment of mammary dysplasia: "If the majority of surgeons were women rather than men, the outlook of the best practical procedure might possibly be different." would have better been omitted, for they convey no idea or information and only reveal the prejudice of the author. The statement that fibroadenoma (of the breast) is "due to" estrogenic stimulation is open to considerable misinterpretation and "dependent upon" might have better expressed the situation as it is understood at the present time.

In the author's attempt to present an orderly arrangement and classification of the extensive material, there was some over-simplification which may result in misunderstanding. An example of this is that of not considering diabetic gangrene in the group of wet gangrene as well as in the dry. Also, the retention of the term "epidermoid carcinoma" is unfortunate because its meaning has become a point of confusion in many circles. More properly, it should be relegated to the same list of outmoded terms in which the author puts "chronic cystic mastitis". Minor errors, such as the misspelling of Halsted's name in two places, can be expected to be remedied in future printings.

The chapter on the thyroid gland is outstanding and gives a clear picture of a subject which is generally confusing. The format is pleasing, the printing is in the popular double column, and the illustrations (many of them new) are numerous and good. The references are well chosen and up to date, with the exception of a few older classics and others of historical interest which have been retained.

Dr. Boyd's writing is lucid, his unique discussions are stimulating, and despite minor inaccuracies as noted, it can be highly recommended as a good working text for undergraduates, even though in the preface it is stated that the book is written expressly for interns and residents.

David T. Petty, M.D.

ABSTRACTS SECTION

LUISADA, ALDO A. (Assoc. Prof. of Medicine; Director, Division of Cardiology). Recent Advances in the Diagnosis of Rheumatic Heart Disease. Amer. J. Med., *17*:781-790, 1954.

The diagnosis of rheumatic heart disease requires recognition of the etiology of the process and evaluation of myocardial, endocardial and pericardial lesions, as well as of possible lingering activity of the rheumatic process. History and physical are important. Myocardial damage may be determined by the electrocardiogram. Various laboratory tests and the clinical picture are of help in the diagnosis of active rheumatic carditis.

Murmurs should be evaluated carefully: (1) because children and adolescents may present systolic murmurs of undetermined nature, possibly innocent, and (2) because rheumatic carditis may cause not only an apical or pulmonic systolic murmur but also an apical mid-diastolic or presystolic murmur. The differential diagnosis between the apical diastolic murmur of mitral stenosis and that of "relative" stenosis caused by carditis is aided by phonocardiography.

The differentiation between "pure" mitral stenosis and mitral insufficiency plus stenosis may be necessary in relation to possible surgical repair of the valve. The following diagnostic methods are briefly reviewed: (1) Physical examination and low frequency trancing, (2) auscultation and phonocardiography, (3) electrocardiography and vectorcardiography, (4) ballistocardiography, (5) pressure tracings of the left atrium, (6) esophagocardiograms, (7) roentgenograms and roentgenkymograms, and (8) electrokymograms. In general, "pure" insufficinecy or stenosis is recognized without difficulty by means of physical data plus electrocardiography, phonocardiography and roentgenology. On the other hand, demonstration of associated mitral insufficiency in a case of mitral stenosis may be difficult and use of the various subsidiary diagnostic methods may be necessary.

A ventricular pressure pattern is transmitted to the left atrium in cases of mitral insufficiency. Esophogocardiography, roentgenkymography, electrokymography, direct measurements of atrial pressure and digital exploration permit recognition of this abnormal pressure wave which causes systolic expansion of the atrium. Electrokymography is the simplest. While it is valuable, it tends to overemphasize the disturbance although calibration and analysis of the tracings may remedy this. Digital exploration tends to underestimate the insufficiency. Therefore, if technical difficulties can be surmounted, pressure measurements with closed chest and no anesthesia may become the most accurate method.

The various technical aids for diagnosis of an associated aortic, pulmonic or tricuspid defect are discussed.

LUISADA, ALDO A. (Assoc. Prof. of Medicine; Director, Division of Cardiology), and MARCUS, EMANUEL (Assistant Professor of Surgery). The Behavior of a Transplanted Heart. Cardiologia, *25*:197-211, 1954.

The function of the transplanted heart was studied in dogs by observation and palpation; by electro- and phonocardiography; by volume tracings; by angiocardiography.

The survival of the organ was studied in normal conditions; after cortisone; after wrapping the heart in amniotic sac or nonirritant cellophane; and following cross-matching of the bloods of animals involved. None of these special procedures prolonged the survival time.

The histological aspect of the transplanted heart was studied after arrest of the organs. It was concluded that an inflammatory process, caused by the immunological reaction of the host, develops within all strata of the organ, from the epicardium to the endocardium.

The transplanted heart reacts to mechanical compression with an increase in rate. This can be explained either through direct increase of excitability of the S-A node by stretching, or through short reflexes.

The reaction of the transplanted heart was studied and compared with that of the recipient's heart following administration of adrenalin, digitalis bodies, or morphine. The transplanted heart is an extremely interesting and sensitive test object for these pharmacological studies.

The transplanted hearts, in general, had a remarkable regularity of action until their beat ceased. This underlines the perturbing effect of autonomic stimuli in cardiac patients.

* * * *

KOPPER, PAUL H. (Assistant Professor of Microbiology and Public Health). The Assimilation of Glucose by Resting Cells of Escherichia Coli. J. of Bacteriology. *67*:507-510, 1954.

Incubation of 18 hour resting cells of a strain of *Escherichia coli* with various nutrient substances for 20 minutes, followed by centrifugation and washing, brought about a subsequent increase in their reducing activity with 2,3,5-triphenyltetrazolium chloride. This is attributed to substrate assimilation.

This effect was most pronounced with glucose but also was noted with a number of salts of carboxylic acids, peptone, and culture broths. Compounds that failed to stimulate bacterial reducing activity were unable to support the growth of the organism in synthetic culture media.

The uptake of glucose by the cells was studied in relation to glucose concentration, time of exposure, oxygenation, concentration of phosphate ions, pH, and temperature.

Under suitable environmental conditions, borate could replace phosphate in inducing glucose

assimilation. Mixtures of equal parts of NaCl and KCl and low concentrations of sodium azide and 2,4-dinitrophenol, respectively, were found to exert a pronounced inhibitory effect on the assimilatory process.

* * * *

FALLER, INGA L., PETTY, DAVID (Associate in Surgery), LAST, JULES H., PASCALE, LUKE R., and BOND, E. E.: A Comparison of the Deuterium Oxide and Antipyrine Dilution Methods for Measuring Total Body Water in Normal and Hydropic Human Subjects. J. of Lab. and Clin. Med. *45*:748-758, 1955.

The deuterium oxide and antipyrine spaces were studied in a large group of adult males. It was found that the deuterium oxide spaces were consistently larger and significantly larger than the antipyrine spaces. It was found that deuterium oxide could be used to measure total body water by oral or intravenous administration with equally reliable results.

In edematous subjects deuterium oxide was found to equilibrate in all body fluids so that it could be used to determine total body water in such subjects. Antipyrine, on the other hand, often failed to equilibrate within twenty-four hours, and it was concluded that its dilution was not a reliable measure of total body water in edematous states.

* * * *

FALLER, INGA L., BOND, E. E., PETTY, DAVID (Associate in Surgery), and PASCALE, LUKE R.: The Use of Urinary Deuterium Oxide Concentrations in a Simple Method for Measuring Total Body Water. J. of Lab. and Clin. Med. *45*:759-764, 1955.

An accurate method for the recovery of urinary deuterium oxide is presented. The total body water in liters was determined by dividing the actual weight of deuterium oxide administered (grams) by the grams of deuterium oxide per liter. The total body waters of thirteen normal male subjects obtained from simultaneous blood serum and urinary deuterium oxide determinations were identical.

There is no difference in the values obtained from urine samples (obtained from three and five hours). The deuterium oxide lost during the period of equilibration is negligible. Equilibration times of deuterium oxide among the various body fluids in edematous subjects are long, and in such subjects the measurement of total body water required many hours with minimum fluid intake, and many samples for analysis.

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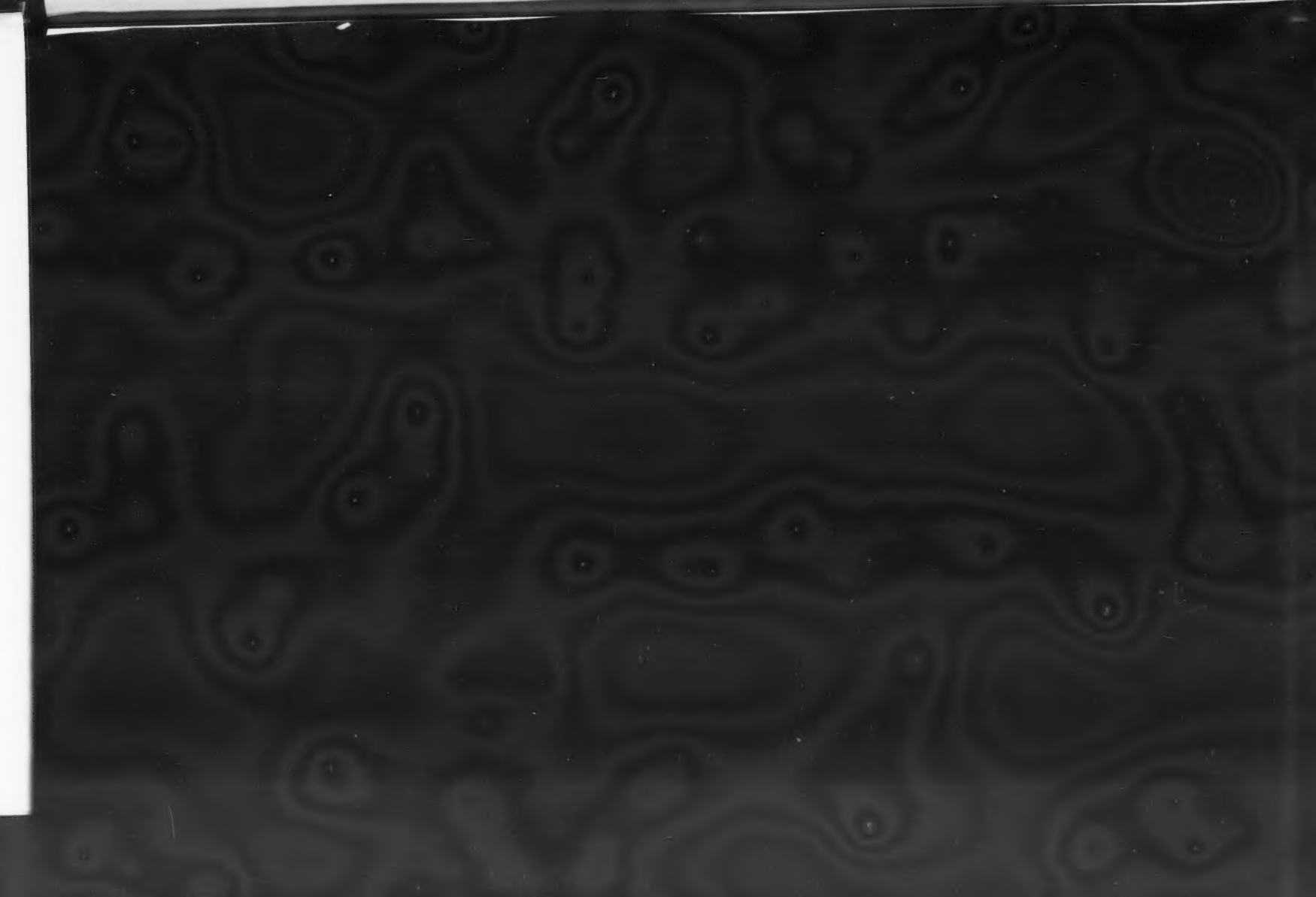

THE CHICAGO MEDICAL SCHOOL

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1955

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